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THE CENTENARY NUMBER

IT is just twenty-five years since THE BRITISH JOURNAL OF SURGERY was founded, and it would seem fitting to introduce this one-hundredth number with a retrospective note.

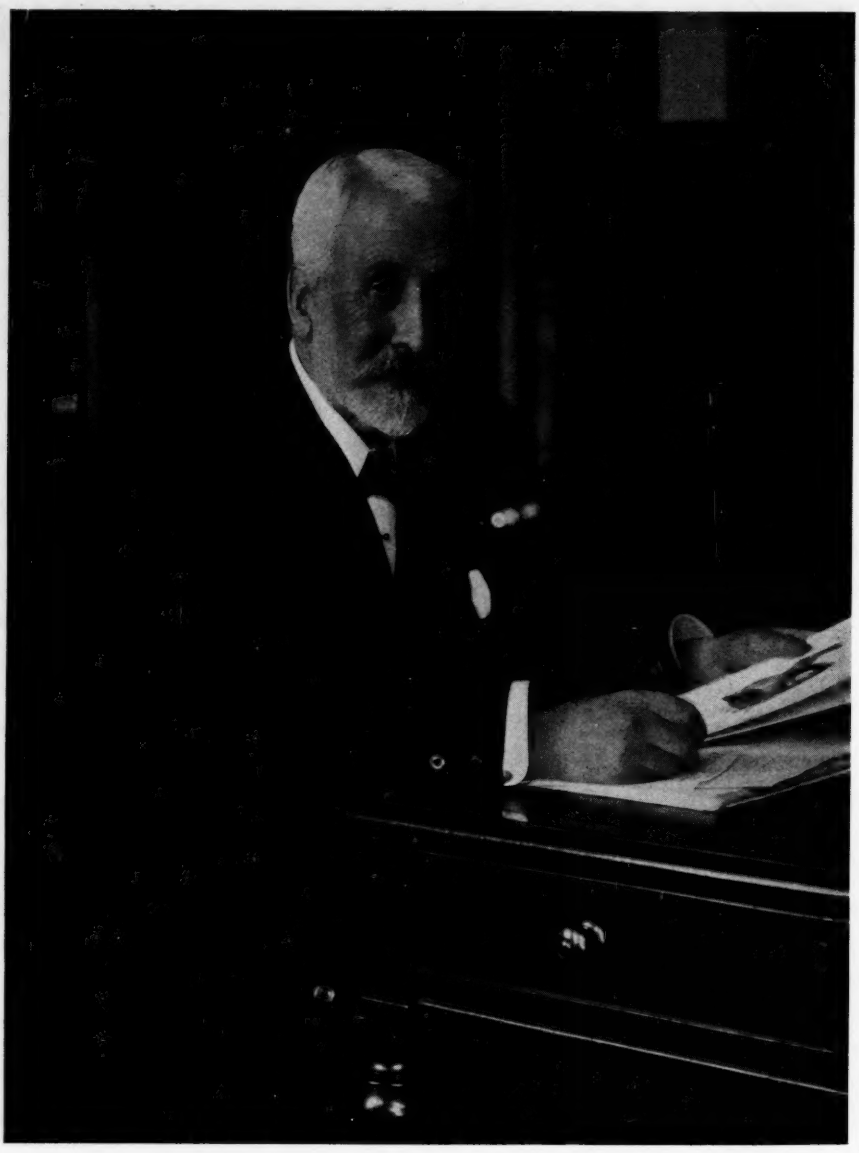
The idea of the JOURNAL arose from a surgical book club in Bristol, when an attempt was made to collect and read the leading representative surgical journals of Europe and America. The fact that no such journal existed representative of British Surgery became painfully obvious. More than one such journal had been started and had failed. The history of these failures showed that lack of success had been due in the main to two causes—they had been purely Metropolitan productions, and had each depended almost entirely on one man.

This matter was discussed with Mr. Hartland S. Wright, the head of one of the first provincial medical publishing houses. By a sad coincidence we have to record in this very number Mr. Wright's death, and we gladly take this opportunity of placing on record the debt the JOURNAL owes to him for his wise advice and practical business support.

From the outset we determined that the JOURNAL must enlist the active support and interest of all surgeons in the British Isles. In particular, we had to secure those great leaders of the British surgical renaissance who worked and taught outside London, e.g., Moynihan, Jones, and Stiles, as well as representative surgeons of all the great London Hospitals. When this representative Committee had been secured, Messrs. John Wright & Sons Ltd. undertook to publish the JOURNAL, and assumed all the financial responsibility involved.

When Moynihan agreed to captain our enterprise, it became comparatively easy to obtain the rest of the team. The first Sub-Committee of Management consisted of Sir Berkeley Moynihan (Chairman), Sir D'Arcy Power, Messrs. Burghard, Corner, English, Fagge, Sampson Handley, and Hey Groves. Moynihan remained our active and inspiring leader until his death in 1936, and we have placed on record in the Royal College of Surgeons an appreciation of our debt to him.

Now that we have attained a vigorous maturity, we can afford to look back on those early days of struggle for existence when we were hard put to it to find



MR. HARTLAND S. WRIGHT

Chairman of John Wright & Sons Ltd.
Born 1848 Died 1938

His faith, enterprise, and experience enabled this Journal to be founded in 1913

material for our pages. Two factors greatly helped us. First, the unwearied help and encouragement we received from Sir D'Arcy Power, who contributed one of his delightful biographical or historical articles to almost every issue. Second, the Great War not only helped to give us material in the form of articles on Military and Naval Surgery, but brought us into contact with American, French, and Belgian surgeons, who came to recognize the JOURNAL as the leading British exposition of surgery.

In 1920 the Association of Surgeons of Great Britain and Ireland was formed under the leadership of Sir Berkeley Moynihan, and served to strengthen the JOURNAL by a re-affirmation of those principles of co-ordination between the leaders of surgical progress which had been the mainspring of its origin.

Now that the JOURNAL has been established for twenty-five years and has taken its place as the leading publication on British Surgery, we look forward with confidence to the future, knowing that we can rely upon the co-operation of all our colleagues in the British Isles and the overseas Dominions.

THE EVOLUTION AND DEVELOPMENT OF SURGICAL INSTRUMENTS

By C. J. S. THOMPSON, M.B.E., Ph.D.

HON. CURATOR OF THE HISTORICAL COLLECTION, MUSEUM OF THE ROYAL COLLEGE OF SURGEONS OF ENGLAND

IV. THE TREPAN

Trepanning is one of the earliest surgical operations known, and there is ample evidence to show that it was practised by primitive man in prehistoric times. The scraping of a hole in the cranium at that remote period was probably carried out with the object of relieving pains in the head or epilepsy, and at the same time providing an exit for the disease demon or evil spirit and to allow its escape (*Fig.* 511).

The operation is still practised among the primitive tribes of New England and New Ireland, and in some parts of northern Africa, while it was formerly common among the Bushmen of Australia. It was usually carried out by

Fig. 511.—Prehistoric trepanned skull of the neolithic period. (*From "Mémoire de Joseph de Baye".*)

scraping an aperture of varying size with a sharp piece of flint or obsidian, after which a bandage of banana fibre was applied over the seat of the operation.

In the western hemisphere, trepanning was practised at an early period by the ancient Incas of Peru, who performed it with great dexterity. It is said that from 5 to 6 per cent of the ancient skulls excavated in Peru show that trepanning was resorted to, not only for therapeutic reasons to alleviate the effects of injury or cure disease, but also to obtain amulets to be worn as charms or to let evil spirits out or to allow good ones to come in. The Incas preferred square openings, and the operation is still practised by native medicine men among some tribes in South America in much the same manner as it was carried out in ancient times (*Fig.* 512).* The patient's head was held tightly between the surgeon's knees—the former reclining and the latter sitting. An incision was then made in the scalp and a section

* Skulls trepanned in a manner similar to that employed by the ancient Incas have recently been excavated at Lachish in Palestine. They are said to date from about 700 B.C.

of bone, approximately an inch square, removed from the skull. The instrument employed was a sharp piece of flint or hardened copper with a rough edge, which

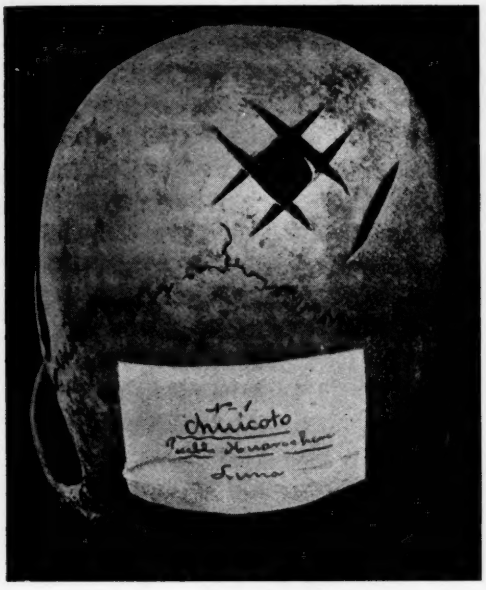


FIG. 512.—Ancient trepanned Peruvian skull. (*From "Mémoire de MacGee".*)

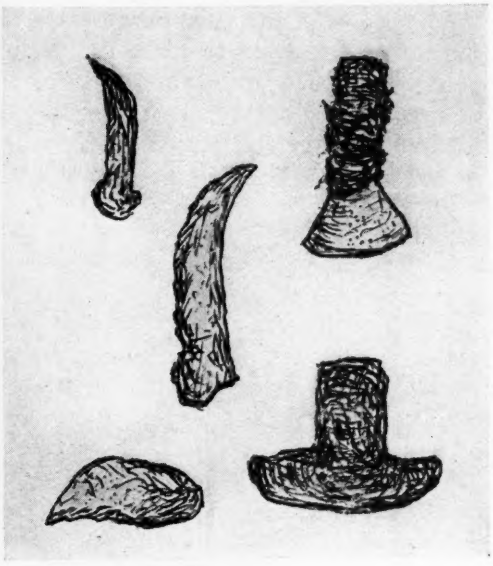


FIG. 513.—Stone and copper instruments used by the ancient Incas in trepanning operations.

was used by rubbing the edge backwards and forwards along the bone (*Fig.* 513). Sudden penetration was prevented by the increasing thickness of the instrument away from its cutting edge. Four grooves were made crossing each other at right angles, and, when they were sufficiently deep, the resulting square of bone was prized out of its bed. Sometimes the aperture was closed with a plate made from shell or other substance, although cases have been found in which the trepan opening was filled with a perfectly fitting diaphragm of lead.

The round trepan openings found in ancient skulls in other parts of the world were usually made by drilling a number of small holes in a circle, or by cutting a circular groove round the piece of bone to be removed, and then breaking down the intervening portions; or in other cases simply by scraping the bone with a piece of flint or stone.

In western civilization we find that trepanning was practised freely in the time

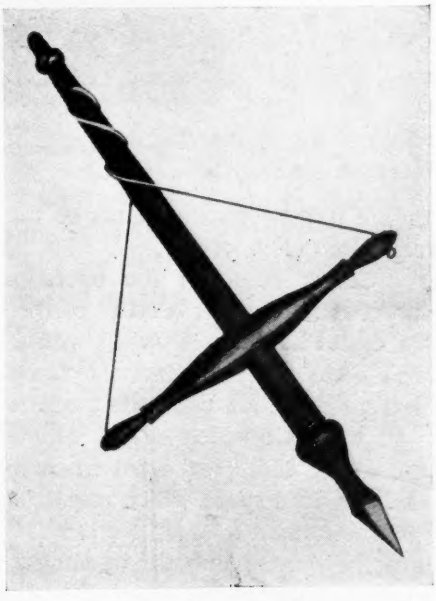


FIG. 514.—The *terebra* used by the early Greek surgeons.

of Hippocrates (*c.* 400 B.C.), and later during the periods of Celsus, Galen, and others. The instruments employed by the early Greek surgeons varied, and included the *terebra* or drill, chiefly used for making the circle of holes round the depressed bone, which was operated by a thong round the centre or on a cross beam (*Fig.* 514). For the same purpose, the trepanon, operated by a bow like a drill, was sometimes employed to make the perforations, and the interspaces between each hole were broken up by the scalpel or lenticular so that the roundel of bone could be removed. Another instrument was the *prion charactos* or *terebra serrata*, which consisted of a conical piece of metal with a circular serrated edge and a centre pin. It had a cylindrical handle several inches long, which was operated by rapidly

FIG. 515.—Trepanning operation upon the skull, from an Oxford manuscript.

rolling it between the palms of the hands. This was a prototype of the modern trephine. (*Fig.* 515.)

Celsus describes the operation and the instruments used by the Roman surgeons in the first century of the Christian era. He tells us that the trepan was "a round hollow instrument with a serrated edge furnished at its centre with a pin which was also surrounded by an interior circle". Of the *terebra*, or wimble, he states, "there are two sorts; one is like that used by a carpenter, the other having a larger blade which begins with a sharp point, then suddenly becomes broader, and above that part again insensibly narrow all the way up. If the disease is confined to a space which might be covered by the trepan or *modiolus* (*Fig.* 516, B) that instrument is to be preferred. If it be caries, the centre pin of the instrument must be pushed into the opening; if blackness, a small depression must be made with the angle of the chisel to receive the pin, so that the trepan may not slip while we are turning it round. It is to be rotated like a wimble, by means of a strap.

The pressure must be so regulated that the instrument may perforate and yet be turned round."

He then tells us how the Roman surgeon solved the problem of the centre pin, which is necessary until the toothed portion had begun to bite. "When the trepan has made for itself a channel," he says, "the centre pin must be removed and the instrument worked by itself; then when by the appearance of the dust we recognize sound bone, the trepan must be withdrawn. If the wimble be used, here too the dust indicates to us how far the wimble ought to penetrate. In whichever way the circle has been made, with the chisel laid flat with the corrupted bone, each superior lamine must be shaved off till nothing but sound bone be left."

He further describes another kind of *terebra*, consisting of a strong copper plate slightly curved backwards and polished on its outward surface; "with this

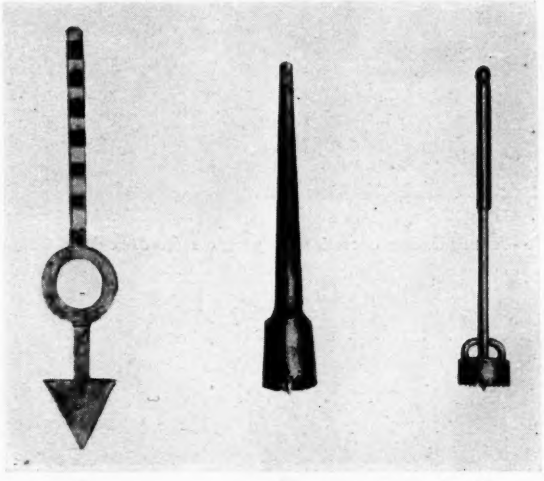


FIG. 516.—A, The spear-headed *incisoria* employed by Arab surgeons, and described by Albucasis; B, The *modiolus* described by Celsus; C, A trepan similar to the modiolus, depicted by Wryghtson in 1350.

surface next the brain it is from time to time put under that part which is to be removed by the chisel, and if it encounters its edge it arrests its further progress."

"Wool steeped in oil and vinegar was applied as a dressing, and in process of time flesh grows from the bone itself and fills up the cavity left after the operation."

In the twelfth century the Arab surgeons were practising trepanning, and we learn from Albucasis that they employed a borer with a sharp point and an instrument terminating with a spear-like head called an *incisoria*, for drilling the holes round the piece of bone to be removed as already described (*Fig.* 516, A). Lanfranc, of Milan, who wrote at the close of the thirteenth century, alludes to "a trepane wich the brayn scalle schal be trepaned with". John Wryghtson, in 1350, depicts an instrument similar in every way to the Roman *modiolus*, which was operated by rolling the shaft between the palms of the hands (*Fig.* 516, C). In the fifteenth century, Jerome Brunswyke describes instruments he calls 'treppanes,' terminating with a long gimlet-like screw, for making small holes in the skull.

When Andrea della Croce began to teach in Venice about 1560, he employed several types of instruments for trepanning, which, judging from the illustrations

in his works, appears to have been an operation commonly practised at the time. He used a brace and drill-stock, to which the circular saw or a sharp perforator was fixed with a screw. (*Fig.* 517.)

FIG. 517.—A trepanning operation in progress (Andrea della Croce, 1573).

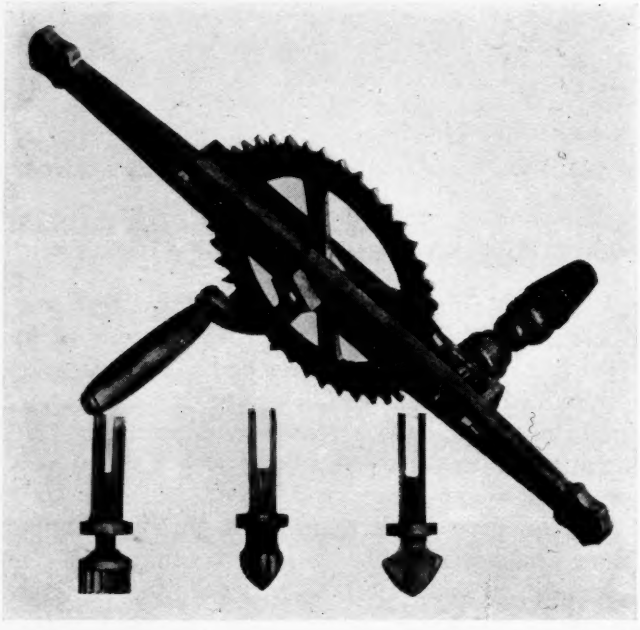


FIG. 518.—The mechanical trepan introduced by Matthia Narvatio in 1575.

The first innovation in the trepan was introduced towards the end of the century, when in 1575 Matthia Narvatio, of Antwerp, invented a mechanical instrument which was operated by a cog-wheel turned by the hand. This was

connected with another wheel, which when rotated actuated a circular saw which cut into the bone, but the instrument was so heavy and clumsy it does not appear to have come into general use (*Fig.* 518).

To Fabricius Aquapendente (1537–1619) is attributed the invention of the trephine, so-called from its triangular shape.* Savigny, in commenting on the instrument, says, " the name trephine is so-called from the triangular form it acquires by the horizontal position of the handle, in contradistinction to the trepan in which the head or crown is affixed to a frame or brace similar to and used in the manner of the carpenter's wimble " (*Fig.* 519). To Fabricius is also attributed the invention of the circular saw with shoulders, with the object of preventing the instrument from sinking down into the membranes of the brain.

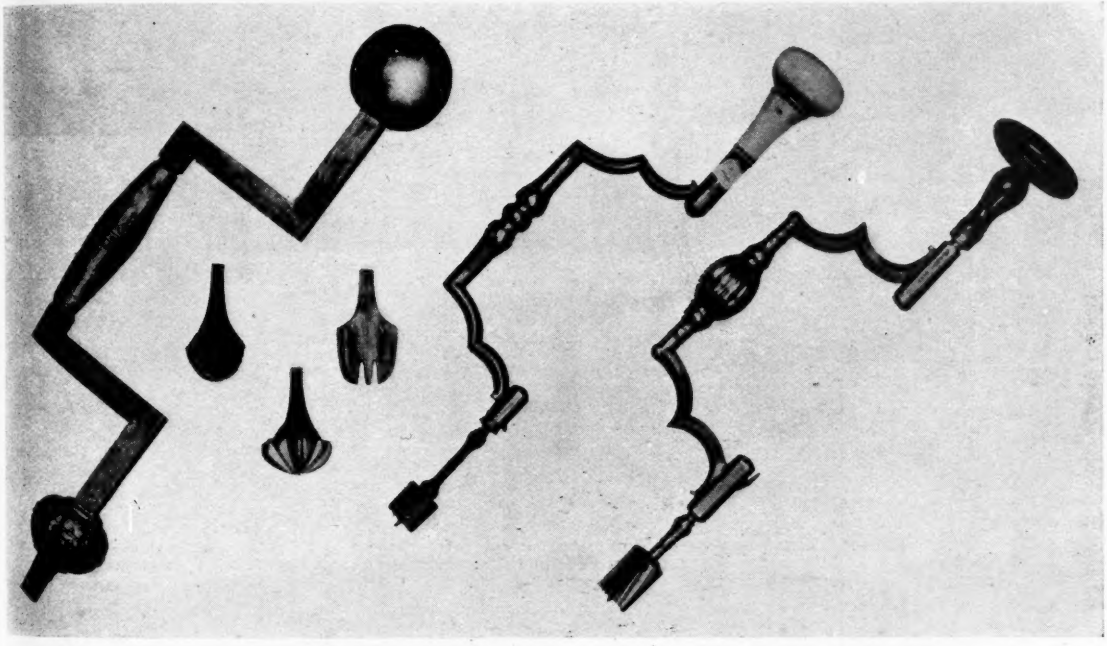


FIG. 519.—Types of brace and trepans in use from the sixteenth to the eighteenth century.

Ambroise Paré (1517–1590), who often operated on the battle-field, employed the brace or drill-stock with a binding screw to fix the saw, the drum of which was straight and smooth, but with a shoulder similar to that recommended by Fabricius. He describes in his work on surgery " trepanes or round saws for cutting out a circular piece of bone with a sharp-pointed nail in the centre projecting beyond the teeth." He also figures a trephine with a transverse handle of metal similar to the instrument introduced by Sharp two centuries later (*Fig.* 520, B).

In 1639 John Woodall, a surgeon of St. Bartholomew's and author of *The Surgeon's Mate*, described his " trafine as an implement of my owne composing ", being probably unaware that Fabricius had preceded him.

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* FABRICIUS AB AQUAPENDENTE, *Opera Chirurgica*, 1628, Frankfurt. In the Leyden edition, 1723, is a figure of the trypana of Fabricius with a cross handle.

Scultetus in his *Chyrurgeon's Store House*, describes what he terms as a 'male' and 'female' trepan, the former having a centre pin and the latter without. "Before we use the females," he writes, "we must make some print upon the skull with the male so that the females may stand the faster upon it. Now for to trepan the skull the Chyrurgian must have at hand at least three trepans exactly equal one to the other; one male and two females, so that he may oft-times change them." He describes "the scraping tool called a lenticular and the levitor or elevator to bring the cut bone forth." He also figures a trephine, "an iron instrument having three sorts of trepans", and an instrument called the *triploides* for raising depressions of the skull in place of the levitor. (*Fig.* 521.)

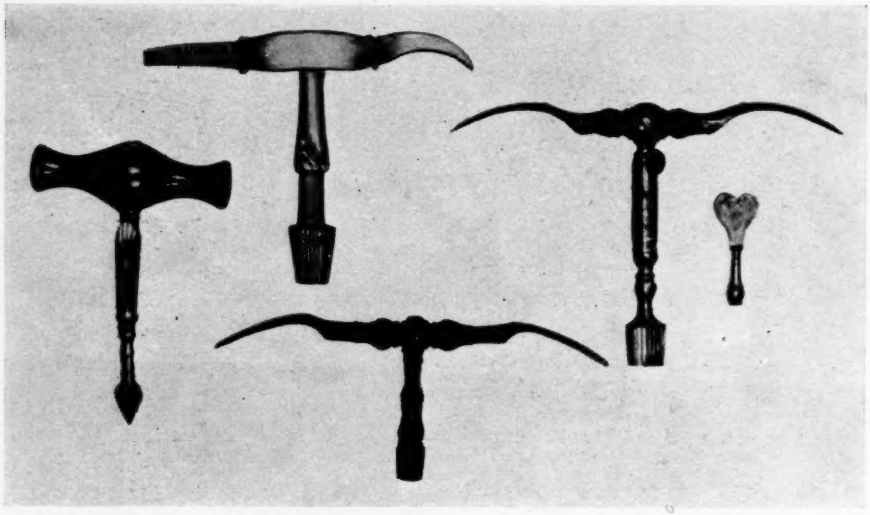

FIG. 520.—A, The *trepan perforatif* and *trepan piramide* of Garengeot (1725); B, Type of trephine used in 1579; C, Perforator introduced by Petit in 1779; D, Sharp's trephine and key for removing centre-pin (1729).

During the eighteenth century several alterations were suggested in the trephine, and in 1725 Garengeot first described his *trepan perforatif* and *trepan piramide* (*Fig.* 520, A).

Fourteen years later, Samuel Sharp, of Guy's, a pupil of Cheselden's, introduced his trephine, which had a wide transverse handle of steel, the extremities of which were roughened for use as elevators. He claimed to be the first to use a trephine with a cylindrical instead of a conical crown-saw, the centre-pin of which could be removed by means of a key (*Fig.* 520, D).

In 1743, Heister described his conical crown-saw, and stated "the moderns have a method of fastening the crown on the trepan otherwise than by screwing, but this is my way." He also introduced an exfoliative trepan, the handle and saw of which was fashioned from one piece of steel.

In 1779, Petit described his perforator, which had a steel drill-head, smooth and bevelled, for drill edges, which he claimed to be an improvement on Sharp's on account of its being shorter; and in 1782, Brambilla figured a trephine very similar in shape to the one introduced by Sharp.

At the close of the eighteenth century, Savigny, writing in 1798, says, "at this time, the trepan used with a brace or wimble is now wholly laid aside in this country." He describes the type of trephine (*Fig.* 523,B) then in use as "a cylindrical saw with a smooth drum, the barrel of which is inserted into the handle direct. The pin has fine teeth with no gaps, and runs in a slot in the barrel, being regulated by a binding-screw and plate. The teeth are perpendicular, which formation is claimed to be an important advantage to the operator."

Then came Benjamin Bell, who introduced his improved trephine in 1801. It had a wooden cross-bar, into the middle of which was screwed a steel key-barrel with a spring grip made to grasp a slot cut

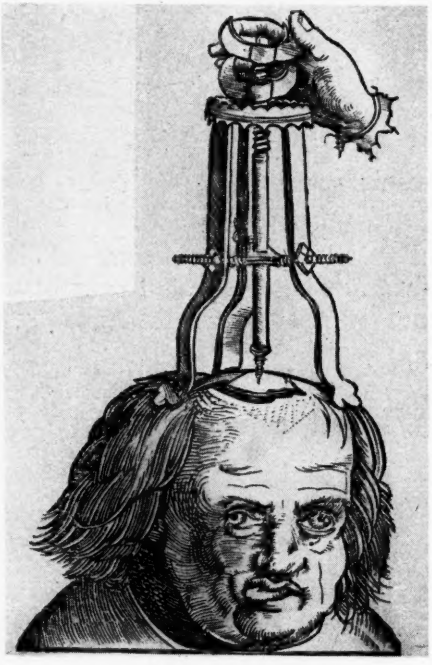

FIG. 521.—The *triploides* (1632).

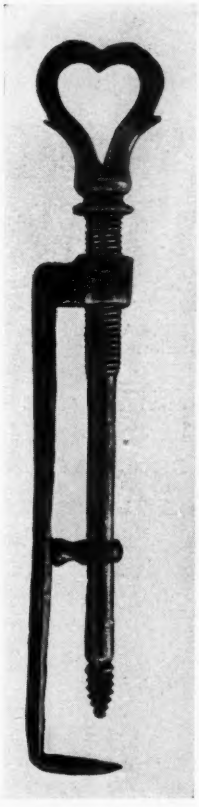

FIG. 522.—An early eighteenth-century instrument for raising depressed bone. (*From the Historical Collection, Royal College of Surgeons.*)

in the key end of the trephine saw. The saw itself had a cylindrical smooth drum with long straight teeth, but, unlike others, these were arranged in three series of nine, and thus three gaps were left to let out the bone dust. This was the forerunner of the 'windows' in the modern instruments. The prismatic was controlled by a thumb or binding screw and plate, which worked in a slot on the side of the brass barrel (*Fig.* 523, A).

Rudtorffer, of Vienna, in 1817 suggested yet another alteration in the form of a screw button in the shaft to raise or lower the pin, and his trephine had a smooth drum (*Fig.* 523, C).

Since the middle of the last century little alteration has taken place in the construction of the trephine.

In 1852, Henry Lee introduced a trephine for necrosis nodes, and successfully removed an abscess of the tibia with it. It was later used by Sir James Paget, Brodie, and Fergusson.

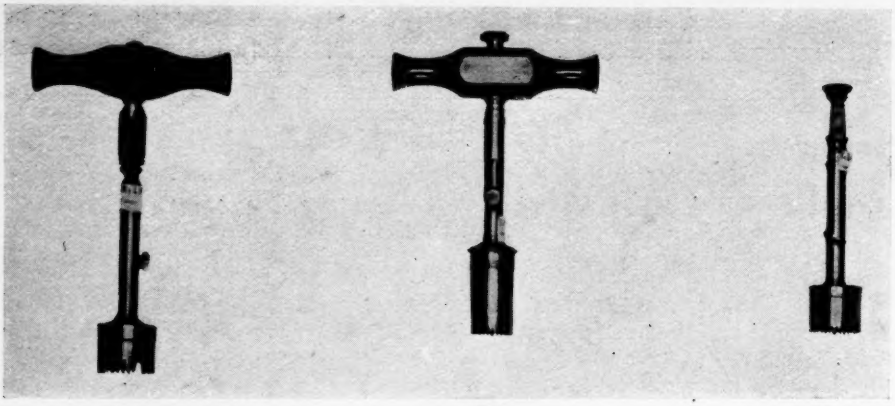

FIG. 523.—A, Trephine introduced by Benjamin Bell in 1801; B, Savigny's trephine (1798); C, Rudtorffer's trephine, with smooth drum and screw button in shaft to raise or lower the pin.

Towards the close of the century there was a return to some of the earlier types of instruments. In 1889, Evans introduced a mechanical rotatory trephine, and also mechanical drill-stocks to be used with drills or trephines, and a pattern

FIG. 524.—Walsham's trephine with crutch handle (1890). This type was used by Sir Victor Horsley.

FIG. 525.—A trephine that belonged to Lister in 1850.

with an Archimedian screw-action appeared in the instrument-makers' catalogues. Macewen's brace was but an adaptation of the early wimble, and Roger's 'skull plough' a reversion to primitive times.

A little later, Walsham introduced a trephine with a crutch-shaped handle, a type which was later favoured by Sir Victor Horsley, but since that time no marked change appears to have been made in the instrument employed on the cranium (*Figs.* 524, 525).

PARALYSIS OF THE BLADDER AND ASSOCIATED NEUROLOGICAL SEQUELÆ OF SPINAL ANÆSTHESIA (CAUDA EQUINA SYNDROME)*

By FERGUS R. FERGUSON and KENNETH H. WATKINS, Manchester

The excellent results which are obtained from the use of spinal anæsthesia may be judged from its popularity in practice. From time to time, however, there have appeared in the literature reports of patients who have developed neurological complications—varying from headache, meningism, cranial nerve paralyses, and focal cerebral lesions to radiculomyelitis, cauda equina, and peripheral nerve lesions—occurring immediately after or remotely following the administration of a spinal anæsthetic. The statements in the literature are extremely contradictory as to the frequency with which such nervous sequelæ occur. Jarman[14] gives the incidence of paralysis as one in ten thousand cases. Foss and Schwalm[12] stated that they had never seen the slightest evidence of peripheral neuritis or sensory or motor disturbance following three thousand spinal anæsthetic administrations. They had never seen a case of foot-drop or had patients who suffered from pain in the body or in the legs following anæsthesia. These authors quote Lundy as never having noticed at the Mayo Clinic that sterile procaine hydrochloride administered by the usual technique and in safe doses produced any serious changes in the spinal cord. Lundy, Essex, and Kernohan,[20] however, stated that they had observed a case of " temporary paralysis " from the use of 10 per cent procaine.

On the other hand, Hyslop[13] reported neurological sequelæ in 11 of 2074 cases recorded by various writers; and 7 examples of neurological complications were reported by Brock, Bell, and Davison.[7] There is no doubt about the frequency with which severe and continued headache and also sixth-nerve paralysis occur. Indeed, Blatt,[5] in 1929, collected over 100 cases of the latter following spinal anæsthesia. Loeser[19] believed that an inflammatory syndrome affecting isolated peripheral nerves was more frequent than was realized, having encountered 5 cases in one year. A review of the literature and the personal experiences recorded by Macdonald Critchley[8] suggest that neurological complications occur much more frequently than is commonly supposed.

In this paper are recorded 14 cases which developed a cauda equina syndrome following the administration of a spinal anæsthetic. Thirteen of these cases occurred within a period of twenty months. Only sixteen examples of this lesion were found in an examination of the literature before this period.

This report is published not with the intention of disparaging a very valuable, if not indispensable, form of anæsthesia, but in the hope that the result of these

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* First presented before the Association of Surgeons of Great Britain and Ireland in London, on May 2, 1935. (K. H. W.)

The neurological aspect was reported in a discussion on the neurological sequelæ of spinal anæsthesia at the Neurological Section of the Royal Society of Medicine in March, 1937 (*Proc. Roy. Soc. Med.*, June, 1937). (F. R. F.)

investigations may help to obviate such unfortunate accidents in the future, and to emphasize the necessity for looking for and recognizing the complications of this method of anæsthesia. This point is stressed because of the impression that symptoms such as paræsthesiæ and sphincter disturbance, and signs such as sacral sensory loss and alteration in the tendon-reflexes, resulting from a lesion in the lower part of the vertebral canal and occurring as post-operative complications, are frequently overlooked.

CASE REPORTS

Case 1.—F. J., male, aged 17, was admitted to the Manchester Royal Infirmary on Aug. 18, 1933, suffering from acute appendicitis and spreading peritonitis. Operation was performed under spinal anæsthesia, for which 'heavy' duracaine was employed. Following operation there was absolute retention of urine and incontinence of fæces. Examination: Left ankle-jerk absent. Diminished sensation to pin-prick over the 'saddle' area. Diminished calf tenderness, with hypalgesia of the left leg. Marked fibrillation in the left lower limb and to a less extent in the right. Anal sphincter tone absent.

The patient made a good recovery from the abdominal condition. The urinary retention was treated by intermittent catheterization until the twelfth day, after which urine passed spontaneously.

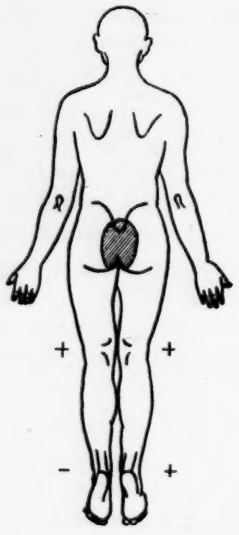

FIG. 526.—*Case* 1. Showing area of diminished sensation to pin-prick.

Sept. 21, 1933.—He reported that he did not now suffer from rectal incontinence, but was constipated. He was urinating two-hourly and twice at night, but not incontinent.

Oct. 5, 1933.—Examination: He passed 180 c.c. urine in a narrow interrupted stream with straining. Residual 150 c.c.

Feb. 15, 1934.—He reported that he was back at work as a labourer. No trouble with the bowels. No frequency of urination and no incontinence. Examination: Anal sphincter tone absent. Residual urine 150 c.c. Urine contained white blood-cells and *B. coli*.

Sept. 29, 1934.—No urinary or bowel symptoms. Reports erections and ejaculations normal. Examination: Anal sphincter tone fair (about 75 per cent). Residual urine 10 c.c. Urine now uninfected.

March 16, 1935.—No urinary or bowel symptoms. Examination: Left ankle-jerk gave the slightest flicker on reinforcement. Anal sphincter tone good (about 100 per cent). Passed 235 c.c. urine in a stream of moderate force and calibre, with some straining and one interruption in the middle. Residual 25 c.c.

Jan. 23, 1936.—No symptoms. Examination: Left ankle-jerk only slightest flicker. Diminished sensation to pin-prick in 'saddle' area, as shown in *Fig.* 526.

Case 2.—S. H., male, aged 44, was admitted to the Manchester Royal Infirmary on Aug. 29, 1933, suffering from perforation of a duodenal ulcer. Immediate operation was performed under 'heavy' duracaine spinal anæsthesia. The patient developed retention of urine immediately after operation and was incontinent of fæces. There was diminished sensation in the 'saddle' area and complete absence of anal sphincter tone. (Condition of tendon reflexes not known—records missing.)

The urinary retention was treated by non-instrumental measures, overflow being assisted by pressure applied to the distended bladder. Bilateral parotitis developed, and on the twelfth day the abdominal wound broke down completely and required resuturing. The patient's condition gradually deteriorated, and he died on the twenty-ninth day.

The method of treating the urinary retention had seemed very satisfactory. Suprapubic expression was not difficult, and the urine remained uninfected. On the twenty-first day the blood-urea was 32 mg. per 100 c.c.

AUTOPSY.—The cause of death was general peritonitis. There was slight dilatation of the right renal pelvis and of both ureters. The bladder was large and the ureteral orifices normal. The vesical orifice was relaxed (it had been hardened by passing a catheter and injecting formalin immediately after death), and the open orifice had a lumen of about 5 mm. diameter. The spinal cord showed no definite abnormality microscopically.

Case 3.—J. G., male, aged 40, was admitted to the Manchester Royal Infirmary on Nov. 16, 1933, and immediate operation was performed for perforated duodenal ulcer, under 'heavy' duracaine spinal anæsthesia. He felt some pain in his back, but not in his legs, when the lumbar puncture needle was introduced. Retention of urine and incontinence of fæces developed after operation. Some urine escaped from the bladder during the first days, but after the fourth day an inlying catheter was employed. Examination: Weakness of the extensors of the right foot and toes. Absence of the right ankle-jerk. Diminished appreciation of pin-prick in the saddle area. Absence of anal sphincter tone.

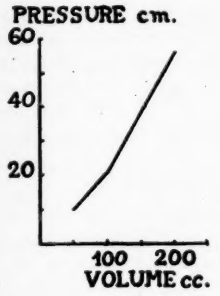

FIG. 527.—*Case* 3. Bladder-pressure curve showing steep gradient of filling characteristic of conus or cauda equina lesions.

Jan. 2, 1934.—He stated that the rectal incontinence was no longer troubling him. He urinated every one and a half hours by day and had to go quickly when he felt the desire or be incontinent. He had to keep a bottle in bed at night owing to incontinence. Examination: Urination was observed to be associated with much straining, which produced a very poor interrupted stream. Following the latter, urine was expressed by suprapubic pressure in a narrow stream which ceased instantly on releasing the pressure.

March 1, 1934.—He had now discarded the urinal and was urinating five or six times both day and night. He only wet his shirt a little during the day, but incontinence was troublesome at night. Examination: He passed urine in a narrow interrupted stream, with straining. Residual 110 c.c. Cystoscopy showed heavy trabeculation and relaxation of the vesical orifice, so that the whole posterior urethra was readily seen with the concave sheath. Bladder 'capacity' 175 c.c.

Aug. 16, 1934.—He can now hold his urine 3 or 4 hours by day, but passes it four times every night. Still slight incontinence at night. Bowels constipated, but not incontinent. Examination: Anal sphincter tone poor. He passed 35 c.c. urine in a narrow interrupted stream. Residual 165 c.c.

Oct. 22, 1934.—Bladder-pressure estimation showed the presence of sensation at 100 c.c. and a very steep curve of filling from the rapid introduction of fluid (*Fig.* 527). The steep curve of filling is characteristic of lesions of the conus or cauda equina.

Oct. 29, 1934.—A cystogram showed numerous small diverticula—no definite evidence of relaxation of the posterior urethra.

March 16, 1935.—He reported that he was doing his full work as a brick setter. Complained of obstinate constipation, but no incontinence. Urination six or seven times during the day, and is rarely incontinent. At night he keeps a bottle in bed on account of incontinence. Examination: Some anal sphincter tone (about 30 per cent). Passed 95 c.c. urine in a narrow stream of poor force, twice interrupted. Residual 175 c.c. Bladder-pressure estimation repeated—confirmed previous observation.

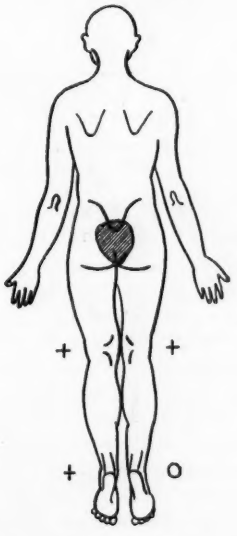

FIG. 528.—*Case* 3. Showing diminished sensation to pin-prick in 'saddle area', with extension to penis and scrotum.

Feb. 5, 1936.—He reported that his only trouble with the bowels was constipation. He urinates every three hours during the day and is not incontinent. Passes urine three times every night, but usually also wets his bed. Examination: He passed 330 c.c. urine in a very narrow stream which was interrupted about six times. There was some straining. Residual 140 c.c. Bladder 'capacity'

(rapid filling) 340 c.c. Urine cloudy. Right ankle-jerk absent. Cannot appreciate the difference between the sharp and blunt ends of a pin in the 'saddle' area. This area of impaired sensation extends on to the scrotum and penis (*Fig.* 528). Anal sphincter tone diminished (perhaps 75 per cent normal).

Case 4.—F. G. H., male, aged 31, was operated on under spinal anæsthesia ('heavy' duracaine) on Dec. 1, 1933, and a lateral anastomosis performed for intestinal adhesions. Before operation there had been no trouble with urination. After operation he developed retention of urine and incontinence of fæces. An inlying catheter was employed. After the latter was removed he passed urine, but had no control over it. On Jan. 2, 1934, the bowel trouble was much improved, but he urinated half-hourly during the day and kept a bottle in bed at night. Examination: Knee-jerks exaggerated, both ankle-jerks active. Impairment of sensation to pin-prick over small 'saddle' area extending partly on to scrotum and penis. Anal sphincter tone considerably reduced (about 50 per cent).

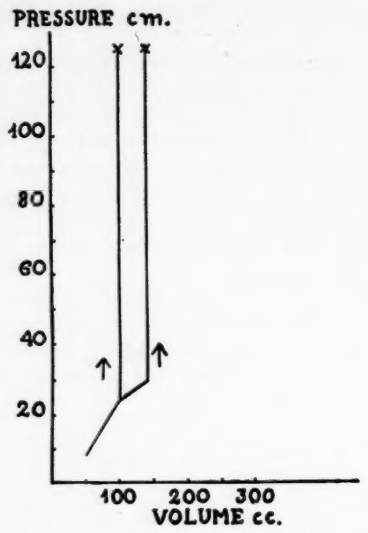

FIG. 529.—*Case* 4. Bladder-pressure curve, showing detrusor contractions.

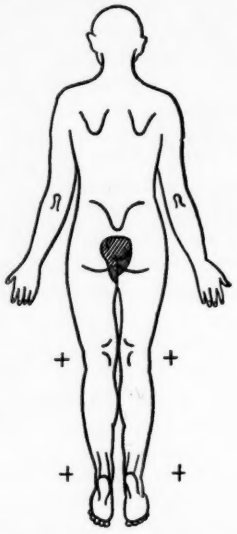

FIG. 530.—*Case* 4. Showing area of diminished sensation.

Jan. 15, 1934.—Examination: He passed 30 c.c. urine with about seven or eight interruptions, the latter coinciding with the intervals between the efforts of straining. Residual 120 c.c.

Nov. 20, 1934.—He reported that he had started work again as a grocer. The bowels were constipated, but he had had no incontinence for a long time. He had to urinate every 1½ to 2 hours, and two to four times at night. Examination: Ankle-jerks both active. Impairment of sensation over 'saddle' area so that he cannot distinguish between blunt and sharp end of a pin. Anal sphincter tone practically normal. He passed 60 c.c. of urine in a narrow stream of poor force with two or three interruptions. Residual 5 c.c. Urine cloudy, containing many white blood-cells, and a few red cells with many bacilli. Bladder-pressure estimations revealed sensation with only 50 c.c. in the bladder and a rather steep type of curve. Definite detrusor contractions were demonstrated, with some escape of fluid around the catheter (*Fig.* 529). These findings indicated recovery of reflex micturition.

March 20, 1935.—He was still troubled with constipation. He could now hold his urine a little longer during the day (occasionally 4 to 5 hours), but gets up two or three times every night. No incontinence. Examination: He passed urine in a splendid stream. No residual.

Jan. 16, 1936.—His constipation was not as troublesome as formerly. He urinates about twelve times during the day and once at night. No incontinence. Examination: Residual 15 c.c. Bladder 'capacity' 150 c.c. Urine crystal clear—no deposit on centrifuging.

March 6, 1937.—He states that rectal sensation is not normal—unable to tell whether flatus or fæces. When he went to the lavatory he could not get rid of all the motion, and would have to go again a few minutes later. If he did not he would be incontinent. Tended to be constipated. He said that he urinated eight to ten times in the day and sometimes once at night. Patient married two years after operation, and states sexual power normal. Examination: Knee-jerks and ankle-jerks present and equal. Definite diminution of sensation to pin-prick, cotton-wool, and hot and cold over the 'saddle' area (*Fig.* 530), with a sharp line of demarcation. Anus a little patulous, but sphincter contraction good—almost normal.

Case 5.—W. B., male, aged 28, underwent an operation on May 11, 1934, for a left inguinal hernia under spinal anæsthesia. (The drug employed is not stated, but inquiry reveals that it was in all probability 'heavy' duracaine.) After operation he developed complete retention of urine and incontinence of fæces. An inlying catheter was employed, and when this was removed after about five days the urine simply dribbled from him. The incontinence subsided after four or five months, and the frequency gradually improved, so that by the end of six months he could hold his urine 4 or 5 hours and was getting up twice at night. The incontinence of fæces troubled him for about $2\frac{1}{2}$ months and there was a good deal of constipation, but this soon subsided. He returned to his work as a riveter after $4\frac{1}{2}$ months.

Oct. 27, 1934.—Examination: He passed 200 c.c. urine in a stream of moderate force and calibre with one interruption. The urine was crystal clear and contained only an occasional epithelial cell. Knee-jerks and ankle-jerks present, but left ankle-jerk not quite as active as right. There was a limited area of impairment of sensation in the 'saddle' region in which pin-prick was recognized but not as sharply as normal (*Fig.* 531). Anal sphincter tone extremely poor.

Nov. 10, 1934.—Residual urine 125 c.c.

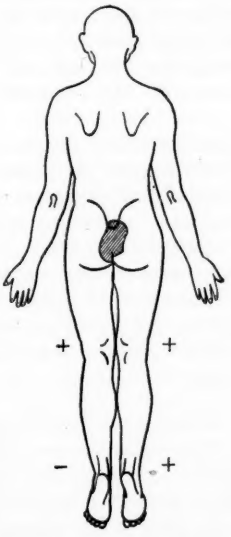

FIG. 531.—*Case* 5. Showing area of impaired sensation.

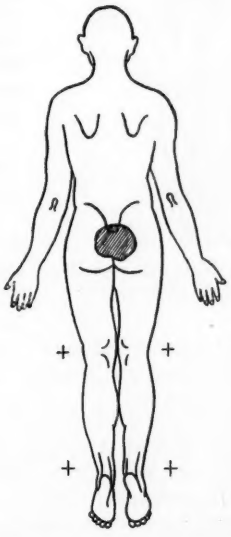

FIG. 532.—*Case* 6. Showing area of impaired sensation.

Case 6.—A. W., a female, aged 20, was operated on for bilateral hallux valgus on June 27, 1934, under 'heavy' duracaine spinal anæsthesia. For a few days after operation she was incontinent of fæces and noticed a numbness about the seat. Incontinence of urine occurred once after operation, after which urination appeared to be normal.

July 1, 1934.—Examination: Diminished sensation to pin-prick in the 'saddle' area. Anal sphincter tone entirely absent. (Ankle-jerks not investigated owing to dressings on feet.)

Nov. 25, 1934.—She reported that the only trouble with her bowels was a tendency to constipation. She stated that urination did not seem to be as free as it was before her operation. She has to strain, and the stream is interrupted. No incontinence. Examination: Residual urine 200 c.c. Urine contained only a few epithelial cells. Knee- and ankle-jerks active. Anal sphincter tone absent.

March 6, 1937.—She stated that she had not the normal feeling of the motions passing, and had marked constipation requiring a purgative every night. (She was not troubled with constipation prior to operation.) There was some hesitancy in urinating. Married two months ago and thinks sexual intercourse normal. Examination: Knee- and ankle-jerks present and equal. Diminished sensation to pin-prick, cotton-wool, and cold in a clearly demarcated area (*Fig.* 532). There is a small trophic ulcer within this area (apparently attributable to cycling, which she took up recently). Anus patulous. Sphincter tone about 30 per cent normal.

Case 7.—M. I., a female, aged 55, was operated on as an emergency for a twisted ovarian cyst, on Aug. 5, 1934. 'Heavy' duracaine was employed and the spinal anæsthetic was given in the sitting position, painlessly. It produced anæsthesia to the costal margin, which lasted 80 minutes. After operation she became incontinent of urine and fæces. Examination: Knee-jerks and ankle-jerks absent. There was dense anæsthesia to pin-prick over the 'saddle' area (*Fig.* 533). Anal sphincter tone absent.

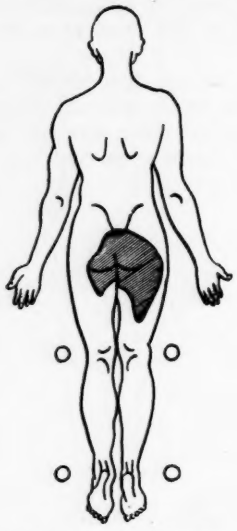

FIG. 533.—*Case* 7. Showing area of anæsthesia to pin-prick.

She had been catheterized once, and as there occurred a rigor on the sixth day an inlying catheter was employed, 71 oz. of heavily infected urine being withdrawn. Subsequently the general condition improved rapidly. She was incontinent whenever the catheter was removed, and as there were pressure sores over the sacrum the catheter was retained. Six and a half weeks after operation she succeeded in passing urine with the assistance of much straining. She passed it every three or four hours during the day, but was incontinent at night. At this time there was still no control over the bowels.

Sept. 26, 1934.—Bladder pressure estimation showed delayed appreciation of sensation (500 c.c.), and a bladder capacity of 1200 c.c. No evidence of detrusor contractions.

Oct. 10, 1934.—She was now urinating every 1 or 2 hours during the day, but at night she was always incontinent. She stated that the urinary stream was interrupted, and after the stream had apparently finished she could always pass some more by straining. She was managing her bowels better, but still had frequent accidents. Examination: Knee-jerks active. Ankle-jerks absent. Dense anæsthesia to pin-prick in the 'saddle' area. Anal sphincter tone absent.

Case 8.—T. H., male, aged 54, was admitted to the Manchester Royal Infirmary on Oct. 11, 1934, suffering from a perforated duodenal ulcer. Operation was performed under spinal anæsthesia, for which 'heavy' duracaine was employed. Retention of urine immediately followed operation. He passed some urine on the third day, but intermittent catheterization had to be continued. It was noted that eleven days after operation he was incontinent both of urine and of fæces. For the next ten days an inlying catheter was employed, after the removal of which he passed urine. Owing to very severe cystitis a suprabic cystostomy for drainage was undertaken on Nov. 8, 1934, for which spinal anæsthesia, 'heavy' duracaine, was again employed.

Nov. 27, 1934.—Examination: Knee-jerks both active. Right ankle-jerk diminished compared with the left. Definite sensory loss over the third, fourth, and fifth sacral segments.

Dec. 20, 1934.—Examination of cerebrospinal fluid is reported as follows: "A moderate number of red cells, but less than 2 leucocytes per cm. Sugar, 0·054 per cent. Proteins, 30 mg. per 100 c.c. Globulin, no increase."

Feb. 10, 1935.—The patient's general condition is very poor, and the cystitis very severe. Examination: Right ankle-jerk still relatively diminished. Slight hypalgesia in fifth right sacral segment and over dorsum of penis.

Feb. 19, 1935.—Patient not incontinent of fæces now. Suprapubic fistula still present. Examination: Anal sphincter tone absent.

This patient died on Feb. 26, 1935. The cause of death was the perforation of a large tuberculous ulcer of the terminal ileum. There was advanced tuberculosis of the lungs and scattered tubercles in the liver, spleen, and kidneys. There was some congestion of the pia-arachnoid, but no degeneration of the spinal cord was evident microscopically.

Case 9.—W. E., male, aged 32, was operated on on Dec. 14, 1934, for right-sided abdominal pain. Appendicectomy was performed under spinal anæsthesia ('heavy' duracaine). The patient states that at the time of operation he was placed on his right side, and the left leg gave a jump when the needle was put into the back, but he did not feel any acute pain. Apparently the analgesia was not complete and a general anæsthetic was given. Following the operation he developed retention of urine, and was treated for ten days by intermittent catheterization. Subsequently he was able to urinate in an interrupted stream by the help of considerable straining, and three weeks after operation he was urinating every hour during the day and getting up two or three times at night. He was troubled with constipation before operation, and this became still worse afterwards, but he never suffered actually from incontinence. At first, after operation, however, he could not retain enemata. Buttocks and sacral region felt numb for the first four or five days after operation.

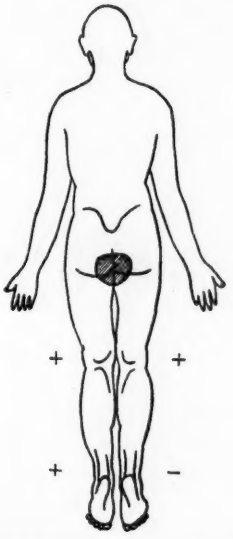

FIG. 534.—*Case* 9. Showing area of diminished sensation to pin-prick.

Dec. 23, 1934.—Examination: Knee-jerks and ankle-jerks present and equal; a small perianal area of diminished sensation to pin-prick (no loss to light touch); anus normal.

Jan. 30, 1935.—He stated he was urinating a little more frequently than normal during the day and usually not at all at night. His main trouble was a considerable degree of urgency. Examination: Knee-jerks active. Both ankle-jerks present, but right not quite as active as left. There is impairment of sensation to pin-prick over the 'saddle' area. Anal sphincter tone almost normal. Residual urine, 210 c.c.

March 1, 1935.—He developed an acute epididymitis on the left side.

June 28, 1935.—He stated that he was not having any trouble with urination apart from some urgency. Bowels very constipated, but control perfect. Residual urine 230 c.c.

Nov. 28, 1935.—He stated that he was not having any trouble with urination and that his only trouble is constipation. Sexual power normal, but was diminished for six months after operation. Examination: Passed 350 c.c. clear urine in a good stream. Residual, 60 c.c.; urine showed no cellular deposit and culture sterile.

March 2, 1937.—Constipation still much more troublesome than before operation. No trouble with urination, though he notices occasional interruption in the flow. Examination: Knee-jerks brisk and equal. Ankle-jerks both present, right less than left. Loss of sensation to pin-prick over fourth and fifth sacral segments, with a sharp line of demarcation (*Fig.* 534). Calf tenderness normal. Anus patulous at rest; good sphincter tone on contraction.

Comment.—In this case the ankle-jerks were equal ten days after operation, but seven weeks later and subsequently the right ankle-jerk was diminished.

Case 10.—G. D. J., male, aged 52, was admitted to the Manchester Royal Infirmary on Jan. 5, 1935, and immediate operation was performed for a perforated gastric ulcer. The patient was placed on the left side for the spinal anæsthetic, and he did not feel any pain apart from the initial prick. There was no difficulty about the procedure; the needle was introduced between the 4th and 5th lumbar vertebræ, and 2 c.c. of 'heavy' duracaine were used. About twenty-four hours after operation urine began to dribble away, the

bladder being very distended. On the fifth day a catheter was passed, and left inlying for a few hours. Incontinence of fæces was present and lasted for several weeks. Blood Wassermann reaction negative.

Jan. 10, 1935.—Examination: Knee-jerks active. Right ankle-jerk active, left diminished. There is impairment of sensation to pin-prick over the 'saddle' area, extending down the back of the left thigh, over the scrotum, and on to the penis. In this area pin-prick is not very sharp, but he can tell the difference between the blunt and sharp ends of the pin (*Fig.* 535). Anal sphincter tone absent.

Jan. 14, 1935.—The urine was heavily blood-stained and infected. The bladder was distended to the umbilicus. Blood-urea, 224 mg./100 c.c. An inlying catheter was therefore employed and intravenous saline given. The general condition rapidly improved.

Feb. 11, 1935.—The catheter failed to evacuate urine from the bladder, although fluid was readily introduced. Nor could he urinate naturally. Suprapubic cystotomy was therefore performed under local anæsthesia, and a large thin membranous slough was removed. This was evidently a 'cast' of the whole bladder wall. The bladder wall seemed a little thickened and the whole of the interior of the bladder seemed to be insensitive to manipulation. The vesical orifice was relaxed.

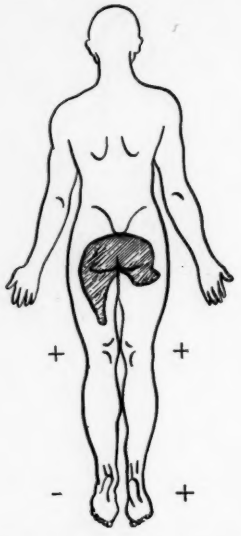

FIG. 535.—*Case* 10. Area of impaired sensation to pin-prick.

March 16, 1935.—Cystoscopy was performed painlessly without any anæsthesia. There was no enlargement of the prostate—either intravesical or intra-urethral—but there was definite early relaxation of the vesical orifice.

April 10, 1935.—He was still entirely incontinent of fæces. At this time the suprapubic tube had been removed and an inlying catheter was employed. Shortly afterwards he began to pass urine naturally.

April 18, 1936.—He stated that he was still wearing a urinal which he had employed ever since his return home. During the night time he wakens about three times to pass urine. There has not been any incontinence of fæces for a long time. He states that he gets good erections, but has not had sexual intercourse since operations. Examination: He passed 80 c.c. of rather turbid urine in an uninterrupted stream of fair force. Residual, 110 c.c. There is some impairment of sensation in the 'saddle' area, but pin-prick feels sharp.

Aug. 31, 1936.—Suprapubic wound broke down, and all urine was escaping from the fistula. Examination: Some loss of sensation to pin-prick on right buttock. Anal sphincter tone good. Cystoscopy—right ureteric orifice appears patulous. Vesical orifice widely relaxed down to verumontanum.

Sept. 8, 1936.—Operation under ether anæsthesia. Suprapubic fistula dissected out and wound resutured.

March 7, 1937.—No bowel trouble. Chief trouble is frequency of urination—six to eight times every night. Examination: Knee-jerks brisk and equal. Ankle-jerks almost equal—slight diminution left side. Right plantar response indefinitely flexor, left plantar response flexor. No sensory loss to pin-prick, but some loss to light touch in the perineal region. Anal sphincter tone almost normal. Anus is dirty and perineal region soiled (though patient of very clean type).

Case 11.—A. H., male, aged 50, was admitted to the Manchester Royal Infirmary on Jan. 6, 1935, suffering from a perforated duodenal ulcer. General condition extremely bad. Blood Wassermann reaction negative. Operation was performed under spinal anæsthesia and continuous intravenous drip infusion employed. The anæsthetic was 'heavy' duracaine (2·4 c.c.) with the patient lying on his right side. The patient did not feel any pain from the needle, which was introduced between the 3rd and 4th lumbar vertebræ. Following operation there was complete retention of urine. At first he was able to retain rectal saline, but later he could not retain either saline or enema.

Jan. 8, 1935 (about 40 hr. after operation).—Examination: Knee-jerks present and equal, ankle-jerks both absent. Dense anæsthesia to pin-prick over the 'saddle' area and down the back of the right thigh and over the scrotum and penis (*Fig.* 536). Anal sphincter tone absent.

About 30 hours after operation 40 oz. of urine were removed by catheterization. Subsequently the bladder was allowed to overflow and expression could be carried out fairly readily, the bladder being distended to midway between the pubes and umbilicus. During this time the urine remained uninfected, but on the sixth day the blood-urea was 204 mg. per 100 c.c. and the general condition very poor. Accordingly an inlying catheter was employed. He showed well-marked improvement, and on the eighth day the blood-urea was 95 mg. per 100 c.c. On the tenth day he collapsed following hæmatemesis and died in a few hours.

AUTOPSY.—There was a large chronic ulcer of the pyloric end of the stomach. The stomach and intestines contained large quantities of fresh and altered blood. There was some trabeculation of the bladder with pseudo-diverticula. No enlargement of the prostate. There was slight congestion of the spinal cord in the lumbar and sacral regions. The sacral nerves showed fine vacuolation of the nerve-fibres, suggestive of recent degeneration.

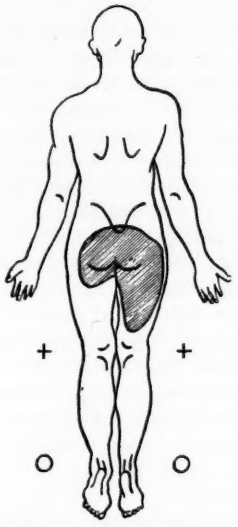

FIG. 536.—*Case* 11. Showing area of dense anæsthesia to pin-prick.

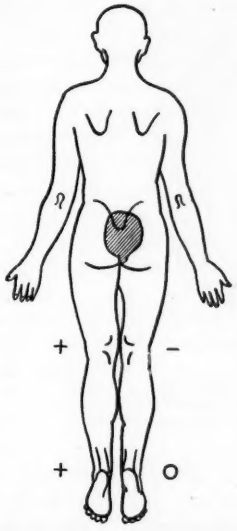

FIG. 537.—*Case* 12. Showing area of sensory loss.

Case 12.—R. G., male, aged 70, was operated on on Feb. 26, 1935, under spinal anæsthesia. Ten stones were removed from the bladder by the suprapubic approach and the prostate was enucleated. The spinal anæsthetic was given with the patient lying on his right side: 2·5 c.c. 'heavy' duracaine were used, diluted to 3·5 c.c. with cerebrospinal fluid. The procedure was painless. It was noticed that for the first ten or fourteen days he had no control over the bowels and that he was frequently soiled. The inlying catheter was removed on March 13, and it was found that he had no control of the urine, which would run from him. There was 14 oz. residual. He also developed a small pressure sore over the sacrum.

March 25, 1935.—He stated that his right leg never seemed to recover properly after the operation, feeling to be weaker and heavier than the opposite limb. He is now able to walk on it, though it seems to drag a little. He says that his bowels are now moving regularly twice a day. He stated that after removal of the catheter he was unable to pass urine himself. Often he would attempt to pass his urine and be quite unsuccessful. A few minutes later he would feel the wet coming on to his legs. There would not be any sensation of the urine passing, and the only intimation he had would be the feeling of the wet on his thigh. At night time it would pass in his sleep, and if he did not keep a bottle in the bed the bedclothes would be wet.

Examination: There is slight wasting of the muscles of the right thigh. All movements of the right lower limb are fairly strongly performed, but he cannot raise it from the bed as strongly as the opposite limb, in which contour and movements appear normal. Knee-jerks—right just present on reinforcement, left active. Ankle-jerks—right absent, left active. Anal sphincter tone absent. There was an area of sensory loss over the 'saddle' area, more extensive over the right side (*Fig.* 537). In this area he could not detect either pin-prick or light touch.

Case 13.—J. B., male, aged 55, was admitted to the Manchester Royal Infirmary on March 31, 1935, and a cholecystostomy was performed for acute cholecystitis under spinal anæsthesia. The patient was placed on his right side for the spinal anæsthetic, and the needle introduced between the 4th and 5th lumbar vertebræ without causing pain: 2·2 c.c. 'heavy' duracaine was injected after diluting with 0·5 c.c. cerebrospinal fluid. About ten minutes later the patient collapsed suddenly, but he recovered sufficiently for the operation to be completed. Blood Wassermann reaction negative.

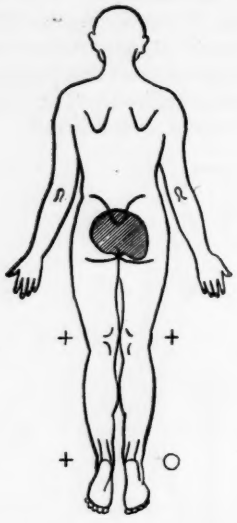

FIG. 538.—*Case* 13. 'Saddle' area of impaired sensation, extending forwards on to scrotum and penis.

After operation he developed retention of urine and incontinence of fæces. He also noticed that he could not feel the passage of a tube for an enema, and that he was not able to retain the latter. The bladder became distended to the umbilicus and was allowed to overflow. Expression was difficult. When on the fifth day the blood-urea was 308 mg. per 100 c.c., he was put on intermittent catheterization, and later an inlying catheter was employed.

April 2, 1935 (less than 48 hr. after operation).—Examination: Knee-jerks active, left ankle-jerk active, right absent. Impairment of sensation to pin-prick over the 'saddle' area and forwards on to the scrotum and penis (*Fig.* 538). He can, however, distinguish between the sharp and blunt ends of a pin. Anal sphincter tone absent.

May 10, 1935.—The urine was passing spontaneously but entirely without control. Incontinence of fæces still present. Catheterization evacuated 200 c.c. urine from the bladder.

May 25, 1935.—Cystoscopy without any anæsthetic was painless. There was moderate trabeculation and one diverticulum and several pseudo-diverticula. There was well-marked relaxation of the vesical orifice, so that the verumontanum was readily seen with a concave sheath.

May 31, 1935.—Cystogram showed a bladder of fairly smooth outline with a diverticulum of small size. No evidence of relaxation of the vesical orifice.

June 19, 1935.—He was passing urine frequently, but evidently without control, and had to keep a bottle in bed. His only knowledge of the urine passing is when it wets him. He can avoid incontinence of fæces only if he is given a bed-pan very quickly.

June 22, 1935.—He passed 150 c.c. urine in an interrupted stream of moderate force, the interruptions coinciding with intervals in the effort of straining. Residual, 60 c.c. Bladder-pressure estimations showed the steep type of curve characteristic of lesions of the conus or cauda equina (*Fig.* 539). Sensation at 125 c.c.

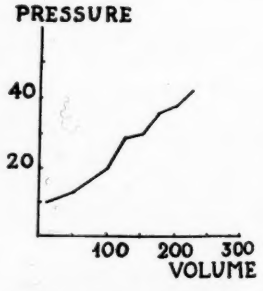

FIG. 539. — Bladder-pressure curve, showing typical steep gradient of cauda equina or conus lesions.

July 4, 1935.—A cystogram showed a small and irregular bladder outline, the diverticulum previously seen, and a 'funnel' urethra (relaxation of the vesical neck).

Sept. 11, 1935.—He was still entirely unable to control his urination either by day or night. He was beginning to get some, but not complete, control of the bowels.

Jan. 8, 1936.—Anal sphincter tone absent. He passed 70 c.c. opalescent urine in an interrupted stream with much straining. Residual, 220 c.c. Capacity (rapid filling), 150 c.c.

March 23, 1937.—Goes to have bowels moved after every meal, otherwise he would be soiled. Urinates every one or two hours during the day, and employs an apparatus for incontinence at night. If he goes longer than two hours in the day, or if he exerts himself, he will almost certainly be incontinent. He wears a large pad of wool and some waterproof in his trousers. Is not married; no sexual intercourse; gets occasional weak erections. Examination: Knee-jerks brisk and equal. Left ankle-jerk normal. Right ankle-jerk markedly diminished. Loss of sensation to pin-prick, cotton-wool, and hot and cold in the 'saddle' area, with a sharp line of demarcation. Definite diminution of calf tenderness on right side. Anal sphincter tone about 40 per cent normal.

Case 14.—W. M., aged 33, was admitted to the Manchester Royal Infirmary on July 20, 1936, suffering from acute appendicitis and early peritonitis. Operation was undertaken under spinal anæsthesia. The patient was placed on his right side, the injection was apparently made between the second and third lumbar vertebræ, and the patient felt no pain apart from the initial prick in the skin. Clear cerebrospinal fluid emerged from the needle, and 1·8 c.c. 'heavy' duracaine, diluted with 1 c.c. of cerebrospinal fluid, was introduced. This gave perfect anæsthesia for the operation.

On the morning after operation the patient complained of a feeling of numbness about the seat, and thirty hours after operation the bladder was found very distended, though the patient was not in discomfort. Catheterization evacuated a large quantity of urine.

July 22 (40 hr. after operation).—Examination: Knee-jerks both active. Right ankle-jerk diminished compared with left. There is loss of sensation to pin-prick over the 'saddle' area, especially the right side, and over the scrotum and partly the penis. In this area he cannot distinguish between the sharp and blunt ends of a pin, and hot and cold was not so easily distinguished as in the normal skin. There is some anal sphincter tone present, but it is very much reduced.

It was noted on the fourth day that he was not able to retain an enema. Intermittent catheterization was continued for about two weeks. For some days he was very ill with pneumonia, but made a good recovery.

Aug. 17, 1936.—He states he has to strain to pass urine and that the stream is interrupted if he stops straining. No frequency and no incontinence. Bowel control perfect. Examination: Both ankle-jerks active. Well-marked diminution of sensation to pin-prick on right side of 'saddle' area. On left side there is only very slight diminution. Anal sphincter tone fairly good, but not quite 100 per cent.

DISCUSSION

THE NATURE OF THE LESION AND ITS COURSE

In these cases there occurred almost immediately following the spinal anæsthetic the following manifestations:—

Retention of urine, succeeded by incontinence.

Incontinence of fæces, with loss of anal sphincter tone.

Impairment of sensation in the 'saddle' area, in some cases also extending down the back of one thigh.

Absence or diminution of one ankle-jerk (occasionally absence of both ankle-jerks and even of the knee-jerks).

It was clear from examination of all these patients that the basic lesion was the same, but that the variations were due to different degrees of affection of the same syndrome.

The bladder symptoms nearly always followed within twenty-four hours of the operation, whereas rectal symptoms were sometimes delayed—in one case for ten days—almost suggesting a progressive lesion. The latter view was also supported by one case in which diminution of an ankle-jerk only became evident subsequent to the first examination made on the tenth day.

The Bladder Disturbance.—Complete urinary retention developed after operation in all cases excepting one female, whose only apparent bladder disturbance was incontinence on one occasion. (She, however, still had 200 c.c. residual five months after operation.) The period of complete retention varied from about one week to a month, and was followed by incontinence of urine. The latter was usually of short duration (a few weeks), but in 2 cases was still present after more than two years. In the early stages of spontaneous urination there was always marked frequency. In 1 case this subsided in about six weeks, in 2 it passed off in six months, and in 4 others it was still troublesome for periods varying from nine months to twenty-seven months after operation. Residual urine persisted for from six to twelve months or more, and in 1 case was still present after twenty-seven months.

The character of the lesion corresponded with that seen following injuries of the conus or cauda equina (Watkins[28]). Thus the act of urination before recovery had occurred was always associated with much straining, the stream was of narrow calibre and poor force, and it was frequently interrupted whenever the patient desisted from his efforts of straining in order to take a deep breath. Cystoscopy undertaken in 4 cases, between two and three and a half months after operation, showed well-marked trabeculation (with diverticula in one case) and relaxation of the vesical orifice. The latter, whilst very definite on cystoscopic evidence, was not usually of sufficient degree to be recognized in a cystogram; in only 1 case did the cystogram show a 'funnel' urethra. The cystograms showed small bladders of irregular outline, and in 2 cases the presence of small diverticula. In 1 case bladder-pressure estimations were undertaken less than two months after operation, and showed a large-capacity bladder with low pressure and delayed appreciation of sensation (500 c.c.). Two cases, at three and eleven months after operation respectively, showed very steep pressure curves, characteristic of conus and cauda equina lesions (*Figs.* 527, 539). In 1 case, twelve months after operation, the pressure estimation showed evidence of powerful detrusor contraction with escape of fluid around the catheter—evidence of recovery of reflex micturition (*Fig.* 529) (Watkins[27]).

The Bowel Disturbance.—The abnormality varied from an inability to recognize when flatus was being passed and loss of normal sensation prior to bowel movement to precipitancy of defæcation and incontinence. The incontinence of fæces lasted less than one month in 6 cases. In 3 cases it lasted about three months, and in 1 about six months. The anal sphincter tone began to return in periods varying from one to eighteen months. In 1 case it had fully recovered in six weeks, in another it was still markedly diminished after thirty-two months.

Sensory Disturbances.—A sensation of numbness—at first constant, then after about a year intermittent—in the 'saddle' area was a universal complaint. This numbness was still present in some of the patients three years after the operation.

Objectively there was sensory loss to light touch, pin-prick, and hot and cold —at first complete, later incomplete (in the 3rd, 4th, and 5th sacral segments, extending in some of the severe cases on to the 2nd sacral segment). The line of demarcation from normal skin was in all cases well defined.

In all but one case the sensory loss has persisted, and is but little changed in extent after eighteen months to three years.

Reflexes.—Usually there was complete absence of one ankle-jerk at the onset, but the alteration in the tendon reflexes varied with the severity of the case. In one mild case all the tendon reflexes were present and equal, but in all the others they were altered—ranging from a diminution of one ankle-jerk, through absence of one or both ankle-jerks, to absence of both ankle-jerks and one knee-jerk, or finally absence of both knee- and ankle-jerks.

The knee-jerks, however, were never diminished or absent unless the ankle-jerks were absent, and they always returned to normal before the Achilles jerks.

In one case the ankle-jerk was still absent after twenty-seven months, and in another it was only just present at the end of twenty-nine months.

The plantar responses were flexor in all cases. In one mild case the bulbo-cavernosus reflex was normal ten days after operation—in other more severe cases this reflex was absent.

The Motor System.—Some weakness, atrophy, and fibrillation of one or both legs—generally more marked below the knees—were present in practically all cases, but they were not prominent compared with the sphincter and sensory disturbances, and the muscular power was almost normal in all cases within two or three months. Marked weakness of the peronei and the extensors of the foot and toes was recorded in one patient.

The Sexual Function.—Very little certain evidence is available in these cases as to the effect on sexual function. In one patient intercourse was satisfactory one month after operation. Another who had also a mild lesion stated that he lost the capacity following operation, but there was recovery of this function in five months. Satisfactory information is lacking with regard to those more severely affected.

There were three deaths in this series, but in none of them could death be attributed directly to the spinal anæsthetic. In all three the operation was for a perforated duodenal ulcer. One died ten days after operation following a severe hæmatemesis. The second died about a month after operation from general peritonitis, and the third about four months after laparotomy with extensive pulmonary tuberculosis and a recent perforation of a tuberculous ulcer in the ileum.

Autopsies were performed in the three cases, but no definite abnormalities were found macroscopically in the nervous system. Sections of the cord showed some increased hyaline changes in the vessel walls and in addition some marginal pallor of the cord in the sacral region, but no other definite changes. Marchi sections were negative. Dr. Susman, who examined the sections carefully, considered that the sacral nerves showed fine vacuolation and evidence of recent degeneration.

THE CAUSE OF THE LESION

It is quite evident that the lesion under discussion is the direct result of the administration of the spinal anæsthetic, since in all cases it appeared immediately following operation. The clinical manifestations of the lesion might be explained either by damage to the lumbosacral region of the cord or to the conus medullaris on the one hand, or to the nerves of the cauda equina on the other, but for several reasons it seems more probable that the maximum damage is to the latter. There are several possible ways in which such damage might occur.

Direct Trauma.—Mingazzini[22] thought that in the case which he reported there might have been direct damage to the conus itself by introducing the needle

at too high a level. This seems to be a possible explanation in some cases, since certain writers are apparently in the habit of injecting at a high level. But it can certainly be excluded as a cause in this series, in which the injections were usually made between the 3rd and 4th, or 4th and 5th, lumbar vertebræ.

It is well known that the lumbar puncture needle may strike one of the nerve-roots of the cauda equina, causing a sudden pain in one or other limb. And it is equally certain that this has happened on many occasions without leading to any untoward symptoms. Of the six patients of this series who were interrogated on this point shortly after operation all definitely stated they felt no pain apart from the first skin prick. One patient, however, stated that his left leg gave a jump when the needle was introduced. It seems inconceivable that the point of the needle should damage several nerves together, as would be necessary to account for the lesion produced. But the strongest argument against trauma is the fact that no sphincter complication or alteration of leg tendon-reflexes has been recorded following simple lumbar puncture or the injection of serum.

Di Pace[24] thought that his case might be explained by the nerves being drawn against the point of the needle by repeated but unsuccessful attempts to aspirate. Lepoutre[18] suggested the formation of a hæmatoma or that the anæsthetic agent had been injected directly into the nerves. It does not, however, seem possible to accept either of these views for those cases in which there has been no technical difficulty and no staining of the cerebrospinal fluid with blood. (There was no technical difficulty and no blood in the 6 cases in which these points were recorded.)

One of us (F. R. F.), in reporting the neurological aspect of these cases before the Neurological Section of the Royal Society of Medicine in March, 1937, put forward the following reasons, which indicated that trauma was not the causative agent in this group :—

1. Tens of thousands of ' diagnostic ' lumbar punctures (some of them with much trauma) have been made without any similar complication. There may be pain in the back, or legs, or headache, but never sphincter complications or loss of the leg tendon-reflexes.

2. There was no evidence of bleeding into the cerebrospinal fluid in those cases of this series in which this point has been noted. Usually the technique has been perfectly simple and straightforward.

3. The lesion is too extensive to be regarded as due to puncture of one, two, or even three nerves.

4. It seems impossible to reconcile the grading of the cases in this series—variations of one lesion—with injury either to a nerve or to hæmorrhage.

5. The post-mortem examinations showed no evidence of hæmorrhage.

6. Lesions also occur in parts of the nervous system distant from the site of injection of the anæsthetic—for example, sixth-nerve palsies.

Inflammatory Reaction.—This would not be expected to cause symptoms so rapidly after the injection. In the three cases which came to autopsy (on 10, 29, and 138 days later respectively) there was no evidence of an inflammatory lesion. A few cases of paralysis due to chronic meningitis have been recorded in the literature. These cases developed extensive paralysis, and those described by Weigeldt[29] and by Delahet and Coureaud[9] did not appear for some time (weeks or months) after the injection. Moreau[23] suggested the possibility of latent syphilis. The Wassermann reaction is only recorded in three of the cases of this series, and in all

of them it was negative. In addition, there was no evidence on careful general neurological examination of a syphilitic lesion of the nervous system.

Toxicity of the Drug.—In discussing whether the lesion in the case which he reported could be due to the toxicity of the reagent itself, di Pace[24] pointed out that not all the nerves which the drug had influenced were affected by the paralysis. This fact was clearly illustrated in the cases of this series in which the analgesia produced might suffice for an upper abdominal operation, and yet the ultimate paralysis was limited to the sacral nerves. Under these circumstances it appeared at first sight unlikely that the paralysis could be attributed to the anæsthetic drug itself. Subsequent investigations, however, led to the belief that only those nerves exposed to the full concentration of the drug are affected.

Of the 14 cases in this series, 13 definitely followed the employment of a heavy solution of the drug known as duracaine. It is probable, though not certain, that this drug was also utilized in the remaining case. Thirteen of these cases occurred in the Manchester Royal Infirmary, 12 within a period of twenty months. During this particular period about 1000 spinal anæsthesias with 'heavy' duracaine were reported in the hospital records. In addition the records include for this period 650 spinal anæsthesias in which the drug is not stated and 60 cases in which a light solution of duracaine was employed. The total number of duracaine anæsthesias must therefore be between 1000 and 1700, giving a rate of incidence for this lesion of between 1 in 90 and 1 in 155 (if the doubtful case is included).

During the period July, 1933, to December, 1935, there were administered at the Manchester Royal Infirmary 1000 stovaine anæsthesias and 300 percaine, apart from those which were unclassified. No case of paralysis has been observed amongst this group (excepting possibly the one in which the drug employed is not entirely certain). Only one further case of paralysis has been observed since the use of duracaine has been almost entirely abandoned. This also followed the employment of 'heavy' duracaine.

CASES REPORTED IN THE LITERATURE

	AUTHOR	DATE	ANÆSTHETIC	NATURE OF OPERATION	DURATION OF COMPLICATION
1	Mingazzini ..	1907	Stovaine	—	—
2	Bilancioni ..	1909	Stovaine	Bilateral inguinal hernia ..	8 weeks
3	Bilancioni ..	1909	Stovaine	Appendicitis	8 weeks
4	Bilancioni ..	1909	Stovaine	Chronic metritis hysterectomy	—
5	Bedeschi ..	1913	?	Ventro-fixation	4 months +
6	Bedeschi ..	1913	?	Appendicitis	4 months +
7	Paul ..	1913	?	Fistula-in-ano	23 months
8	Paul ..	1920	?	Hernia	4 months +
9	di Pace ..	1923	Stovaine	Fistula-in-ano	3 weeks
10	Folly ..	1923	Stovaine	Inguinal hernia	6 months
11	Bazy ..	1927	Stovaine (probably)	Inguinal hernia	3 years +
12	Lepoutre ..	1927	?	Inguinal hernia	3 years +
13	Moreau ..	1927	?	Appendicitis	4 years +
14	Vilar ..	1928	?	Perineorrhaphy	5 years +
15	André-Thomas	1930	Novocain 4 per cent	Imperforate hymen ..	11 years +
16	Boisseau ..	1931	?	Acute intestinal obstruction	22 months +

It is entirely certain, therefore, that duracaine is an exceptionally dangerous drug in this respect. Nevertheless it is not the only drug which can cause this syndrome, and of the 16 cases recorded in the literature 6 were due to stovaine and 1 to novocain, as shown in the table.

The composition of duracaine (heavy solution) has varied. Those solutions most widely employed at the Manchester Royal Infirmary were :—

1. Before the end of 1934 :—

Procaine HCl, 'Planocaine' brand ..	0·1 g. per c.c.
Gliadin	0·00325 g. per c.c.
Glycerin	0·1 g. per c.c.

In 15 per cent ethyl alcohol.

2. After 1934 :—

Procaine HCl, 'Planocaine' brand ..	0·1 g. per c.c.
Gum acacia	0·002 g. per c.c.
Glycerin	0·0416 g. per c.c.

In 15 per cent ethyl alcohol.

It was thought, therefore, that the alcohol and glycerin might be the responsible factors. It seems probable that alcohol may produce a lesion of this type, because Kafer and Sanguinetti[16] recorded an identical lesion following the intrathecal injection of 80 per cent alcohol containing 25 per cent of antipyrin. Moreover, three patients (observed by one of us—K. H. W.) suffering from carcinoma of the rectum, in whom a colostomy had been performed, and who had no disturbance of urination before injection, developed urinary retention lasting for many weeks immediately following intrathecal injection of 1 c.c. or less of absolute alcohol for the relief of pain. On the other side is the fact that quite strong solutions of alcohol can be injected into peripheral nerves without causing permanent damage. Thus Labat and Greene,[17] after injection of alcohol in strengths up to 33 per cent into nerves (paravertebral), found that the resulting weakness lasted only a few hours. According to Dogliotti[10] intrathecal injections of absolute alcohol into dogs produced hypo-æsthesia and motor paralysis which recovered in a few days, but he never observed any interference with the vesical or anal sphincter. Jefferson[15] observed a patient in whom 3 c.c. of 90 per cent ethyl alcohol was injected intrathecally in three doses over a period of a few days. Careful examination subsequently did not reveal any definite abnormality. Similar complications have also followed clinical and experimental spinal anæsthetic injections which did not contain alcohol, e.g., stovaine; and finally, Macdonald and Watkins[21] were not able to produce paralysis in cats by the intrathecal injection of 15 per cent ethyl alcohol with 20 per cent glycerin. It does not, therefore, seem possible that the lesion can be due primarily to the alcohol and/or glycerin.

In an attempt to elucidate this problem, Macdonald and Watkins[21] carried out a series of intrathecal injections in cats. Using solutions of the same concentration as those employed in man, but of greater relative bulk, they were able to reproduce a cauda equina lesion in a considerable proportion of cases with 'heavy' duracaine. Cauda equina lesions could also be produced by the injection of novocain itself, in the same concentration (10 per cent) as that employed in man. Furthermore, the incidence of these lesions was greatly increased by employing a stronger solution

of novocain. There seems to be little doubt, therefore, that the toxic agent is the novocain.

It had at first appeared unlikely that the novocain itself could be the causative factor, because the ultimate paralysis involved only very few of the nerve-roots upon which the drug had acted at the time of the operation. The explanation now seems manifest. The risk of paralysis is greatest in nerve-tissue (nerve-roots, conus medullaris, and sacral region of the cord) which meet the drug in its full concentration. The lesion is therefore maximal in those roots of the cauda equina into the immediate vicinity of which the drug is injected. Before it reaches the more distant parts the concentration has been considerably reduced by admixture with cerebrospinal fluid. A much higher percentage of lesions was produced in cats because the injection of relatively much greater quantities of the novocain solution rendered dilution by cerebrospinal fluid a much slower process. It is an interesting fact that in several cases the extent of sensory impairment was greater on the side on which the patient was lying at the time of injection, and it was on this side also that the ankle-jerk was altered. This may be due to the fact that the solution employed was considerably heavier than the cerebrospinal fluid. It seemed probable that the toxic effect was greater on the nerve-roots of the cauda equina than on the more sensitive spinal cord tissue, since the injection was invariably made at a distance from the latter.

In discussing the cause of the complications of spinal anæsthesia which they reported, Brock, Bell, and Davison[7] state that their relative infrequency makes it necessary to postulate the existence of tissue sensitivity to the cocaine derivatives. It is certain that in this series the tissues of these patients must have been more sensitive than those of the large majority, who did not suffer any such complications. The evidence of the animal experiments, however, leads to the conclusion that this is not in the nature of an idiosyncrasy, but that in the concentration employed the drug itself has a toxicity but little short of that which would produce paralysis in a high percentage of cases.

As has already been stated, the composition of 'heavy' duracaine has varied. Complications have followed when there has been no gliadin or gum acacia present. 'Heavy' duracaine was introduced in order to allow of slow diffusion of the anæsthetic solution in the cerebrospinal fluid and thus permit better control of the extent of the anæsthesia. It is suggested that it is the combination of the procaine with the alcohol and glycerin (and gliadin or gum acacia) which has led to the procaine—in its full concentration—being held too long locally in contact with the nervous tissue close to the site of injection, and therefore being permitted to exert a local toxic effect in susceptible individuals.

SUMMARY

1. Fourteen cases of 'cauda equina syndrome' following spinal anæsthesia ('heavy' duracaine) are reported.

2. 'Heavy' duracaine is a dangerous anæsthetic, and should not be used in its present form.

3. The mode of affection of the bladder, bowel, tendon-reflexes, and sensory disturbances is detailed.

4. The causation and location of the lesion are discussed.

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CARCINOMA OF THE SMALL INTESTINE

By Sir W. I. de C. Wheeler

From time to time the surgeon is confronted with neoplasms of the small intestine or rare forms of ulceration which on clinical examination resemble closely carcinoma of the stomach. The resemblance is most marked when the duodenum or jejunum is involved. The history is one of down-hill progression, unimpeded by treatment of a conservative kind. The story is told of loss of weight, loss of appetite, gastric stasis, intermittent attacks of colicky pain or an indefinite ache in the region of the umbilicus. Secondary anæmia is often obvious, and is associated with occult blood in the stools; the latter is a frequent finding. Visible peristalsis is to be expected in well-established cases, or the hardening of the bowel wall may be felt during a peristaltic wave. This palpable peristalsis is followed by relaxation and softening. The growth may or may not be felt; it is usually movable, and varies in position according to the attitude of the patient. As in the case of other intra-abdominal lesions, palpation is facilitated when the patient stands in the stooping position. The recti muscles are thus relaxed and there is a disposition for growths to be displaced downwards. A case of actinomycosis of the duodenum and jejunum reported by the writer in 1928[1] gave a clinical picture almost identical with that recorded below. It was diagnosed in the first instance as gastric carcinoma. The clinical findings in a second case of actinomycosis of the liver[2] were also misinterpreted. Diagnostic confusion is increased by the presence of multiple lesions such as are frequently found during the course of abdominal operations. Mayo and Nettrour[3] found gall-stones in five cases of malignant disease of the small intestine. A duodenal ulcer was present in two additional cases, and in others diverticulitis, goitre, or tuberculous disease of the spine was present.

Nevertheless, if carcinoma of the small intestine is kept in mind, more especially when clinical findings arouse the suspicion of the presence of a malignant stomach, diagnostic difficulties are by no means insuperable. Much depends on radiological findings. If the stomach is reported to be definitely free from growth, and the colon also is normal, in a case where clinical findings include intermittent attacks of colic and vague abdominal pain, anæmia, and loss of weight and appetite, and occult blood is present, our thoughts should turn to the possibility of carcinoma of the duodenum or jejunum.

Radiological examinations of the small intestine often are not very convincing, but a point of obstruction may be observed, with dilatation of the proximal coils. Sarcoma occurs more frequently than carcinoma, but the age incidence is lower (between 30 and 40 years) and the ileum is most often involved. In addition to the recent communication of Mayo and Nettrour, there is a plethora of references to malignant disease of the small intestine throughout the literature. The writers just mentioned refer to fifteen communications.[4] Raymond Johnson[5] described three cases, two of carcinoma of the jejunum and one of the ileum. It is stated that in none of them was a correct pre-operative diagnosis made. Geoffrey Jefferson[6] described the post-mortem findings in a case of supra-papillary carcinoma of

the duodenum; Bland-Sutton[7] also dealt with the subject of cancer of the small intestine.

Mayo and Nettrour state that seventy-six cases were seen prior to Feb. 1, 1936, and mention the existence of considerably over two hundred cases. These authorities believe that the jejunum is the commonest site, and that carcinoma of the small intestine comprises 0·47 per cent of carcinomata of the gastro-intestinal tract. The prognosis is bad. The operative mortality of carcinoma of the jejunum is 20 per cent, and the average duration of life after operation is 17·6 months.

The purpose of this communication is to put on record a case of carcinoma of the jejunum.

CASE REPORT

The patient, a woman aged 64, was seen in July, 1937. Clinically she appeared to be a case of gastric carcinoma with all the familiar signs and symptoms. A movable mass could be felt just above and to the left of the umbilicus. No visible or palpable peristalsis was observed. The X-ray findings showed a normal stomach and colon, and gave the information that the palpable mass appeared to be outside the stomach and above the colon.

FIG. 540.—Adenocarcinoma of the jejunum. The illustration shows the fatty material and peritoneum removed from the transverse mesocolon to which the growth was adherent.

FIG. 541.—The growth is shown from the side remote from the mesocolon. The constriction is slightly exaggerated.

OPERATION.—The abdomen was opened to the left of the midline over the tumour. The stomach was found to be normal. No pathological lesion was felt in the liver or gall-bladder. The transverse colon and omentum were turned upwards and the growth was demonstrated in the jejunum, about 12 in. below the flexure. It was adherent to the root of the transverse mesocolon. The growth was easily separated from the mesocolon, with a layer of peritoneum and fatty tissue. When controlling the vessels in the latter a heavy clamp was used. There was only slight distension of the proximal jejunum, and obstruction

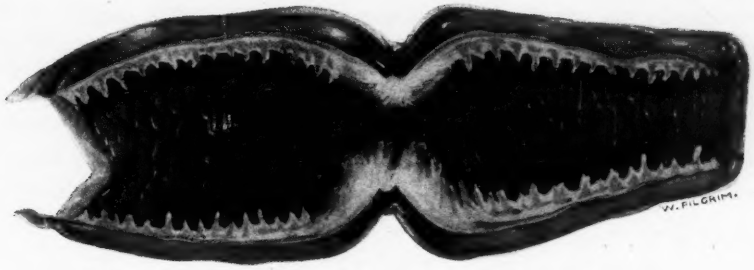

FIG. 542.—The jejunum opened, showing the infiltrating growth. Obstructive symptoms were not a feature of the case.

was not a prominent feature at the site of growth. Eleven inches of the jejunum with the attached growth were resected. End-to-end anastomosis with the aid of Rankin's three-bladed clamp was performed.

SUBSEQUENT PROGRESS.—The patient did well until the third day. She then had a sudden violent pain and collapsed, and showed all the signs of acute peritonitis. It was believed that the anastomosis had given way.

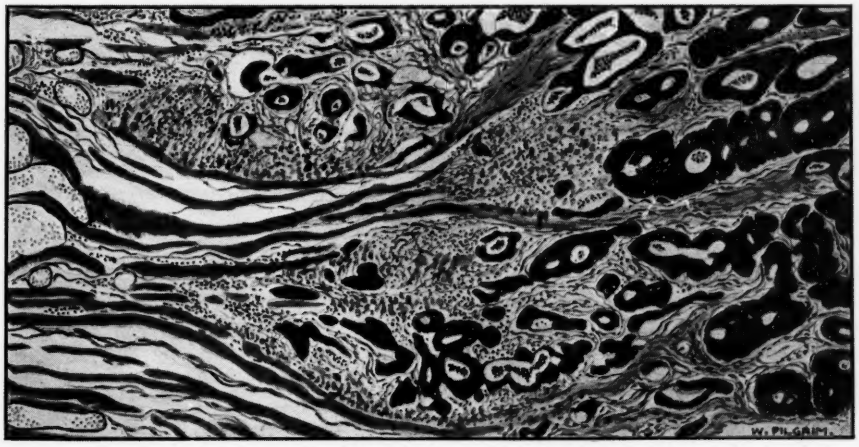

FIG. 543.—Section of growth from the upper jejunum—adenocarcinoma.

AT AUTOPSY.—At the post-mortem a large perforation of the posterior wall of the stomach was found, without signs of previous ulceration. It is believed that the stomach was injured by inclusion of a portion of the posterior wall in the clamp (mentioned above), during the separation of the growth from the mesocolon. No aperture was made in the transverse mesocolon during operation, and it is assumed that the stomach wall was pinched by the clamp controlling the vessels in the fatty tissue. There were no signs of injury when the edges of the aperture in the stomach were examined post mortem.

The accompanying illustrations make clear the appearances of the growth. *Fig.* 540 shows the fatty tissue and peritoneum of the mesocolon removed with the segment of jejunum.

Fig. 541 rather exaggerates the amount of constriction and dilatation of the proximal gut. *Fig.* 542 shows a typical adenocarcinoma such as is found in the distal colon. *Fig.* 543 is a reproduction of a drawing from a microscopical section.

PATHOLOGICAL REPORT (Dr. BOND).—

"1. The anastomosis in the jejunum appeared quite sound, the lumen admitted one finger, there was some hæmorrhage into the neighbouring mesentery. There was a round hole on the posterior wall of the stomach, half way between the curvatures and about one-third of the way from the cardiac orifice. The edges of the perforation were rounded off, but showed no signs of fibrosis or chronic ulceration of the stomach.

"Microscopically the stoma edge is composed of fibrin. The muscle of the wall is rounded off and infiltrated with acute inflammatory cells. There is evidence of hæmorrhage on the peritoneal surface. The mucous membrane shows evidence of chronic gastritis.

"2. Section of growth shows invasion of all coats of the intestine by columnar-cell adenocarcinoma. The lymph-gland sectioned is free from growth."

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AN ENCEPHALOCELE ASSOCIATED WITH HYPERTELORISM AND CLEFT PALATE

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An encephalocele which protrudes into the mouth through a cleft in the palate is rare.

History.—A male infant six weeks old was admitted to the General Infirmary at Leeds on Aug. 17, 1936. Soon after birth the child's mother had noticed a bluish swelling protruding into the roof of his mouth. His lip was partially cleft. Eleven weeks after birth an operation was performed for a hare-lip, which the doctor said was of the median type and extended halfway up the lip. The child was brought to hospital because he had been vomiting persistently for a week. The mother's sister had a child with a left hare-lip and cleft palate.

Physical Examination.—The infant was listless, pale, and grossly underweight. His eyes were abnormally wide apart, and his nose was retroussé. There was a median notch in the upper lip, and leading upwards from this a scar extended halfway to the nostrils. There was a complete cleft of the palate which involved the alveolar margin in the middle line. Just behind the cleft in the alveolus and protruding partially into the cleft, there was to be seen the remains of a vestigial premaxilla, pinkish in colour and covered by ordinary skin. There was no sign of a bony nasal septum. Protruding through the cleft in the hard and soft palate, and situated behind the vestigial premaxilla, was a much larger, maroon-coloured swelling. This was 1½ in. in its long axis, ½ in. in width, and ½ in. in depth. It was attached to the base of the skull by a wide stalk approximately 1 in. in diameter. The tumour was soft and fluctuant, and was noticed to increase in size when the child cried or strained, and upon jugular compression. Pressure applied to the swelling caused the child to vomit. The swelling was almost completely reducible upon firm pressure. It was possible to elicit cross-fluctuation with the anterior fontanelle. (*Figs.* 544–546.)

The skull measured 11 in. from inion to nasion. Its circumference at the widest part was 16½ in. (normal for infant of this age, 16 in.). Stereoscopic radiographs showed that the swelling, which protruded into the mouth, communicated with the interior of the skull through a deficiency of the anterior part of the sphenoid.

Operation.—On Nov. 4, 5 c.c. of clear fluid were aspirated from the swelling, but almost immediately the sac filled again. The fluid was found to contain 9 lymphocytes and 3 red cells per c.mm. and 120 mg. of protein per 100 c.c.

On Dec. 8, under light ether anæsthesia, after reduction of as much of the contents of the sac as was possible by manual compression from outside the sac, the neck of the swelling was transfixed and ligatured. The patient became dyspnœic and blue twenty-four hours after the operation, and died soon afterwards.

Histological Examination of the Tumour (Dr. J. Hart-Mercer).—It was covered for the most part by squamous epithelium, although in many places the

epithelium had been replaced by a fibro-purulent crust. Beneath the surface there was first a layer of circular plain muscle, and then a layer of longitudinal plain muscle, and deeper still, a layer of circularly arranged fibrous tissue. The proximal

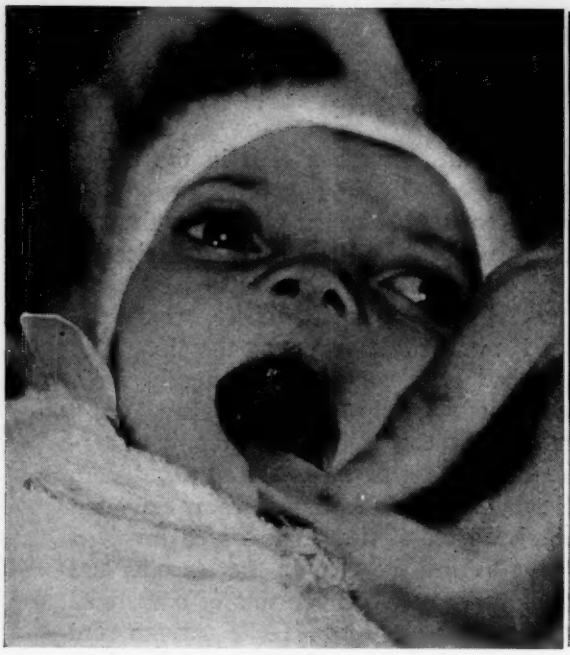

FIG. 544.—Showing the encephalocele protruding into the roof of the mouth, and the abnormally wide separation of the eyes.

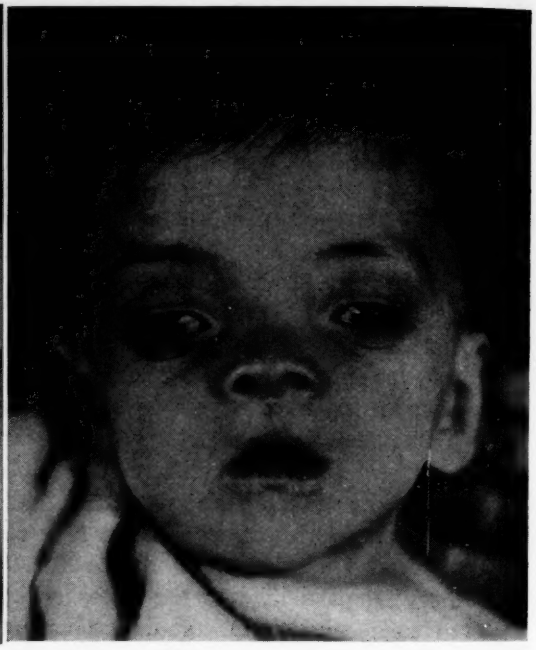

FIG. 545.—Showing the median notch in the upper lip, and the scar above.

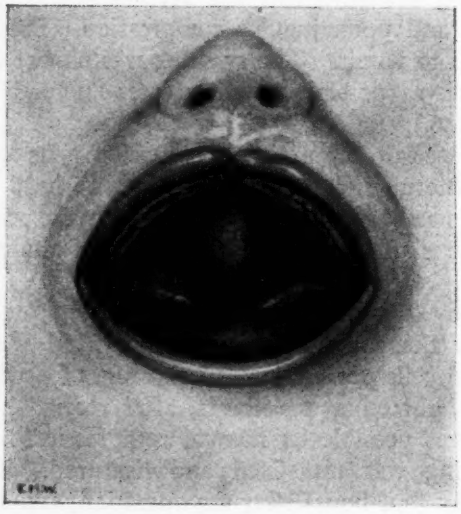

FIG. 546.—Showing the encephalocele protruding through the cleft in the palate. The vestigial premaxilla is seen just between the margins of the cleft alveolus. The median notch in the lip can be seen, with the scar above it.

portion of the tumour was composed of brain substance associated with a mass of tissue, the cells of which resembled those of the anterior lobe of the pituitary body.

AUTOPSY.—The post-mortem examination showed that there was a deficiency of the anterior part of the body of the sphenoid and the vertical plate of the ethmoid. The defect was heart-shaped, and measured 1 in. in the antero-posterior axis, and 1½ in. from side to side in its widest part. It was in direct continuity with the pituitary fossa. The defect contained an evagination of the dura and pia arachnoid, and included a prolongation of brain matter from the region of the floor of the third ventricle. The pituitary body was closely associated with the neck. The optic nerves were lateral to the posterior part of the neck, but were separate from the encephalocele. No definite evidence of meningitis was noted. The cause of death was collapse of the lungs and bronchopneumonia. (*Figs.* 547–549.)

Commentary.—The primary defect which allowed the formation of the encephalocele in this case was probably the incomplete development of the body of the sphenoid bone. This was associated with persistence of the craniopharyngeal canal and ocular hypertelorism.

Rathke[1] was the first to describe a small irregular depression of the pharyngeal mucous membrane near the under surface of the body of the sphenoid in certain mammalian embryos. The depression appeared early in fœtal life, some time before the development of the palate. It extended, in many cases, through the craniopharyngeal canal in the body of the sphenoid into the sella turcica. It is now generally believed to give origin to the anterior lobe of the pituitary body. Obliteration of the craniopharyngeal canal starts near the pharyngeal extremity, and usually takes place early in fœtal life. Kölliker[2] corroborated the presence of this diverticulum in chicks and human embryos of from 4 to 6 weeks. Waldeyer[3] found a fully formed canal in 17 per cent of anthropoid apes and in 10 per cent of human fœtuses and newborn infants. Landzert,[4] in the skulls of newborn infants which he examined at St. Petersburg Foundling Hospital, found also that the craniopharyngeal canal was patent in 10 per cent of cases.

Encephaloceles associated with such canals were described by Klinkosch[5] and Breschet.[6] Fridolin[7] described a case in which a patent craniopharyngeal canal was associated with a hare-lip and a right-sided cleft palate. Bitot[8] described a similar case in which a median hare-lip was present with a craniopharyngeal canal. Lichtenberg[9] reported a case in which a "large bluish-red looking tumour, of the size of a small fist, was hanging out of the mouth, overlapping the chin, and resting with its base on the sternum". The patient had been born with a median hare-lip and cleft-palate, but "handling or even pressing the tumour neither diminished the size of it nor affected the child". After passing a ligature as high as possible round the pedicle, it was cut across, and reddish fluid escaped into the mouth. The child died three days later. At autopsy the tumour was found to contain hypophysial tissue, and the pedicle was seen to pass through a canal in the sphenoid immediately in front of the sella turcica. Dr. Lichtenberg considered it to be a "fair specimen of an encephalo-meningocele". Rippmann,[10] Sonnenberg,[11] and Windle[12] described teratomata protruding through clefts in the palate and associated with patent craniopharyngeal canals.

Mild degrees of ocular hypertelorism are not uncommon in cases of cleft palate. Greig[13], in 1924, was the first to describe ocular hypertelorism. It is a

congenital and often familial deformity in which there is excessive width between the eyes. The nose is retroussé and has a widened bridge. He believed that hypertelorism was primarily due to abnormal development of the sphenoid bone at about the eighth week of fœtal life. It is characterized by overgrowth of the lesser wing and deficient growth of the greater wing. This leads to widening of the pituitary fossa and cribriform plate of the ethmoid, and to increased width between the orbital fossæ.

FIG. 547.—Showing the encephalocele after removing the vault of the skull and elevating the frontal lobes of the brain at autopsy.

FIG. 548.—Showing the defect in the sphenoid at autopsy, after removing the encephalocele which protruded into it. The back of the tongue is seen in the floor of the defect.

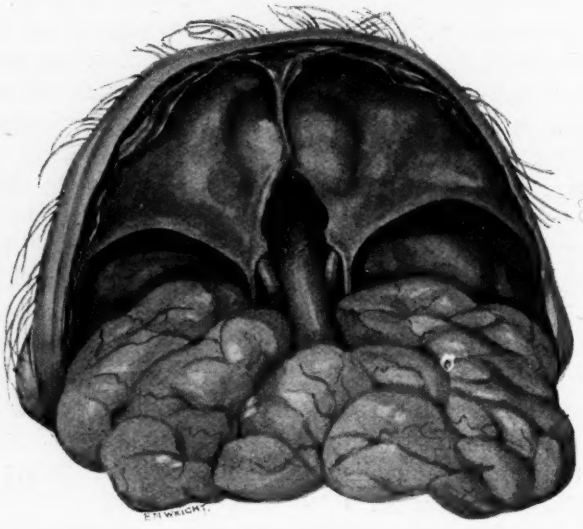

FIG. 549.—Showing the view of the encephalocele from above after the frontal lobes of the brain had been lifted back during the autopsy.

In the case described it can be seen from the photograph of the child's face that the eyes are abnormally far apart and that the nose is retroussé and has a wide bridge (*Figs.* 544, 545)

FIG. 550.—Showing lateral view of the skull, with defect in region of anterior part of the body of the sphenoid, and the faint shadow of the encephalocele protruding into the mouth.

FIG. 551.—Showing lateral view of the skull of a normal child of about the same age for comparison.

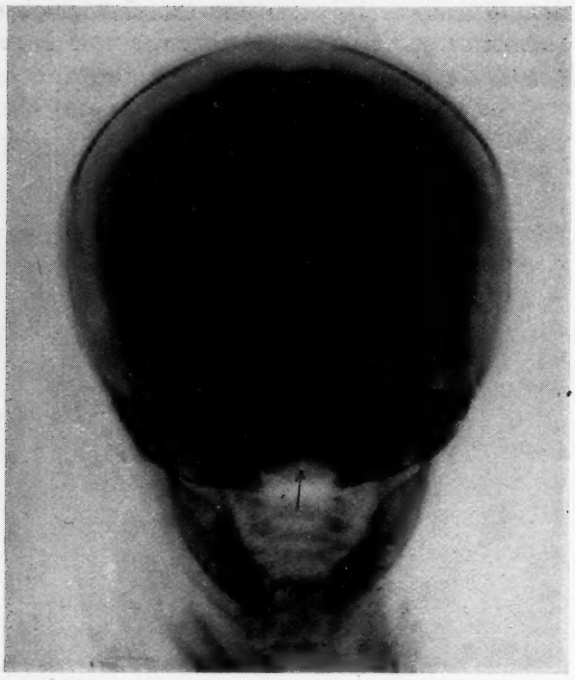

FIG. 552.—Antero-posterior view of the skull, showing deficiency of the base in the midline, the absence of the bony nasal septum, and the deficiency of the horizontal plates of the maxillæ. The faint shadow of the encephalocele is seen.

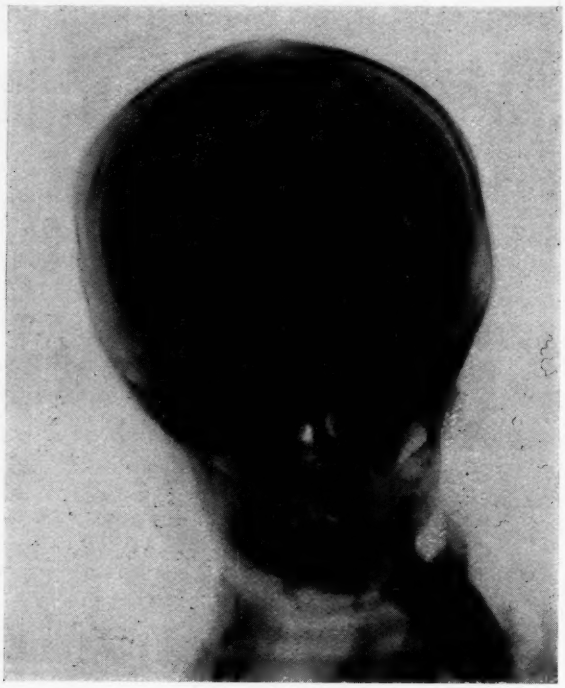

FIG. 553.—Antero-posterior view of the skull of a normal child of about the same age for comparison

Widening of the cribriform plate and the increased distance between the orbital fossæ can be clearly seen in the photograph of the interior of the base of the skull (*Figs.* 547, 548).

Added, however, to this evidence of hypertelorism, there was a defect also in the median part of the body of the sphenoid, homologous with a greatly exaggerated craniopharyngeal canal. Through this defect the encephalocele protruded.

Berry and Legg[14] describe five types of median hare-lip. In the fifth and most extensive type, an extreme cleft extends along the whole fronto-nasal process. They showed a photograph of a patient in whom there was an extensive facial cleft involving the palate, lip, and nose. This was associated with a meningocele protruding through the midline of the forehead near the root of the nose.

Nager[15] described three types of meningocele which protrude into the nasal cavity: (1) *Naso-orbital* associated with a tumour at the root of the nose and on the forehead. (2) *Naso-orbital* associated with protrusion of the eyeball. (3) *Naso-ethmoidal*, resembling a nasal polyp, but being attached to the septum and the roof of the nose.

The important points in the recognition of a nasal meningocele were summarized by Hallermann[16]: (1) The mucous membrane of the septum is continuous over the tumour. (2) Compressibility. (3) Increase in size when the intracranial pressure is raised by jugular compression, or when the patient cries or coughs. (4) Widening of the root of the nose, the space between the eyes being increased. (5) Associated defects of the skull or facial development, e.g., facial cleft, epicanthus (and, we may insert, cleft palate and hare-lip).

To these may also be added: (6) Cross-fluctuation between the swelling and the anterior fontanelle.

Confirmatory evidence is obtained by: (7) Needle puncture, when there is an escape of cerebrospinal fluid in a larger amount than would correspond to the size of the tumour. (8) *Stereoscopic X-ray examination*, and by comparison with radiographs of a patient of similar age, bone defects of the sphenoid, ethmoid, and frontal regions may be seen. (*Figs.* 550–553.)

The prognosis is grave in all basal meningoceles treated by operation. But successful removal of a spheno-orbital meningocele by a transfrontal approach, using a supra-orbital bone-flap, has been described in a girl of sixteen years by Dandy,[17] and in a boy of nine years by Urrets Zavalia et al.[18] A bone-graft, taken from the skull in the flap, was used in each case to occlude the defect. Dandy split the bone in the frontal flap with a chisel. The outer table was used to occlude the defect in the base of the skull, and the inner table was replaced in the flap.

Nearly all cases treated by operation through the mouth or nose have proved fatal. Meningitis has been the commonest cause of death. (Lichtenberg,[9] Kondring,[19] van Duyse,[20] Luz,[21] Hallermann.[16])

Summary.—A case is described in which there was a basal encephalocele which protruded through a cleft in the palate. This was associated with persistence of the craniopharyngeal canal, ocular hypertelorism, and a median type of hare-lip.

The frequent relationship between hypertelorism and cleft palate is noted, and the methods of diagnosis and treatment of basal meningo-encephaloceles are discussed.

My thanks are due to Mr. L. R. Braithwaite and Professor C. W. Vining for permission to publish this case.

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CHRONIC LYMPHŒDEMA*

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The term 'chronic lymphœdema', confessedly non-committal, is used to define a condition of persistent œdematous thickening of a part, usually the lower extremity. This thickening is always present, but varies according to rest and exercise. It affects solely the skin and subcutaneous tissue and does not extend beneath the deep fascia. The lesion may produce an enormous increase in the girth of a limb, and although pitting may be produced by firm pressure the swelling shows the characters of a solid œdema. The overlying skin is of normal colour, not cyanotic, but is liable to show transient red patches of inflammation, and in severe cases may show small vesicles from which lymph freely exudes. The symptoms are aching, made worse by exertion, together with a sense of weight and disability in the heavy limb. There is inevitable limitation in movement of the joints. Varieties of the condition are often described under the names of Milroy's disease and elephantiasis.

Although lymphœdema presents a certain superficial resemblance to the common 'white leg', and may in fact share some features of its pathology, the two conditions must be regarded as clinically separate. Phlegmasia alba dolens has an acute onset, usually puerperal or post-operative, and shows evidence of thrombophlebitis in the thigh or calf. It leaves as its residue a thickened leg, very irritable on exertion, but differs from lymphœdema by its tendency to duskiness, small varices, and by its unfortunate liability to ulceration.

Whilst the pathology of 'white leg' is not understood, it is probable that an element of lymphatic obstruction is present in addition to the venous block. This view is confirmed by the experience of Jennings (1933), who explored Hunter's canal in two cases and found its contents so matted by fibrous tissue that their separation was very difficult.

CLASSIFICATION

In our clinic at the Manchester Royal Infirmary we have had the opportunity to study a large number of cases of chronic lymphœdema. After even a short experience it is evident that, in spite of a general superficial resemblance, the cases fall into groups and that such grouping probably corresponds to differences in aetiology.

Some writers, among them Allen (1934), divide the cases into non-inflammatory and inflammatory, but in view of our ignorance of aetiology the value of any such division is doubtful. We prefer a classification which is based on history and

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clinical features, and would suggest a provisional grouping as follows: (1) Milroy's hereditary œdema; (2) The congenital type; (3) The obstructive type; (4) Post-traumatic œdema; (5) The spontaneous type.

1. Milroy's Hereditary Oedema.—Although the name of Milroy (1892) is often used for other forms of lymphœdema, its use should be restricted to the type of case which Milroy depicted in his classical paper entitled "An Undescribed Variety of Hereditary Œdema". The type which Milroy described would appear to be one of great rarity. His patient, a man of good physique, aged 31 years, suffered from a chronic œdema of both legs to the level of the knees. The condition was stationary and had been present since birth.

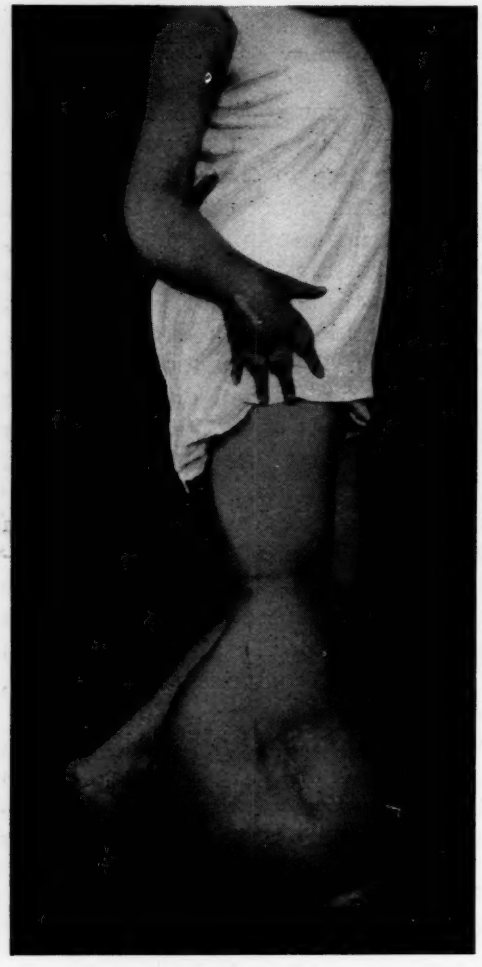

FIG. 554.—*Case* 2. Lymphœdema of congenital origin in a woman aged 23.

Milroy gives a genealogical tree of this patient to the sixth generation, and states that in the six generations there were in all 97 individuals, of whom no less than 22 had suffered from chronic œdema. Of those the sex of which was known, 12 were male and 7 female.

From a suggestion made by Osler it seemed to Milroy that the cases might be related to angioneurotic œdema, but he decided against this view on clinical grounds. A study of Milroy's paper suggests, we think, that the condition he described is more likely to have been a diffuse lymphatic hygroma of congenital origin. The familial transmission of such angiomatous mishaps is, of course, well known.

2. The Congenital Type.—The two following cases are selected as typical of this group:—

Case 1.—A boy of four years was seen with massive œdema confined to the right foot and leg. The lesion extended to the level of the knee, but during the ten years in which he has been under observation the trouble has remained stationary over its original area. A quite adequate Kondoléon operation had been done at the age of three years by another surgeon, but had produced no benefit.

Case 2.—A woman of 23 years was seen with an enormous lymphœdema of the right leg (*Fig.* 554). The disease was also present in the distal portion of the right arm. A clear history was not available, but she thought "it had been there as long as she could remember". Neither could any history be obtained of a somewhat limited and unsuccessful Kondoléon operation. The distribution in the arm and leg of the same side suggests a congenital origin.

We have seen a number of cases of this type in the feet and legs, and on the dorsum of the trunk. This form appears to be a diffuse lymphatic hygroma analogous to the type which is occasionally seen in the neck of infants (cystic hygroma or hydrocele colli). It no doubt represents a congenital aberration in the development of lymphatic vessels analogous to that which results, in the case of capillaries and venules, in venous nævi, and like these it may be localized or diffuse.

3. The Obstructive Type.—In these patients the cause of the œdema is a capillary lymphangitis of widespread nature due in many cases to streptococcal infection and followed by a diffuse peri-lymphatic fibrosis. The condition is seen after frank streptococcal infections, e.g., erysipelas, and is best known in relation to filarial infestations in man. Here it would appear more likely that the resulting elephantiasis is due to the inflammatory reaction rather than to mechanical obstruction by the parasites themselves.

In this group also there fall those œdemas of the arm which are secondary to massive involvement of the axilla by new growths, often with superadded sepsis. A similar condition is seen as the result of post-radiation fibrosis.

We have recently encountered an unfortunate example of the obstructive type in a boy of twelve, who suffers from a huge elephantiasis of the penis and scrotum which is due to a diffuse, hard, matted condition of the horizontal set of inguinal glands in both groins; so far no cause has been found.

Many of the patients in this obstructive class show, from time to time, exacerbations during which areas of redness appear on the affected limb, and the condition is complicated by some rise of temperature—the so-called 'elephantiasic fever'.

The lymphatic obstruction to which cases of this group are due must be diffuse and widespread if it is to cause the disease. Some observers have dismissed obstruction because they have ligatured the main lymphatics of a limb without any harmful result. These writers appear to ignore the widespread anastomoses which are a feature of the lymphatic system. Moreover, there is a constant interchange of fluid between lymphatics and capillaries. It is, for example, well known that the main thoracic duct may be ligatured at the root of the neck without incident.

4. The Post-traumatic Oedemas.—With the exception of an extensive article by Hill (1936), this condition is scarcely mentioned in English writings, but a very considerable literature by Continental authors is available since the first description by Secretan (1901).

The condition may be illustrated by three examples which we have recently seen:—

Case 3.—Male, aged 25 years, chauffeur, struck the back of the left hand a smart blow with a spanner. There was no break of the skin, but considerable swelling appeared at the site of the injury. This did not resolve in the usual way, and when seen three years after the accident there was a prominent, firm, elastic swelling over the back of the left hand, extending to a line 3 in. above the wrist, where it quite abruptly ceased. The fingers were swollen and the hand rendered almost useless by the limitation of movement.

Case 4.—Female, aged 19 years, machinist, struck the dorsum of the right foot against a steel lever on the floor two years ago. The injury was not severe, and she continued at her work. The same night she noticed a swelling on the dorsum of the foot. This swelling persisted and has steadily increased; it now involves the greater part of the dorsum of the foot and extends 2 in. above the ankle-joint, where it shades off imperceptibly into the normal.

Case 5.—Male, aged 23 years, clerk, received a kick on the left external malleolus at football one year ago; it was not very severe; he played out the game, but noticed a swelling the same evening. The swelling did not resolve, but there has spread from the site of injury a chronic lymphœdema which now extends half-way up the left thigh.

Secretan's cases were all similar to *Case* 3, but his paper is open to criticism in that the length of history of his cases is short. He does not mention any upward spread of the lesion. Vulliet (1902) describes similar cases.

In the majority of the recorded instances the injury is a blunt trauma of no great severity and without open lesion of the tissues, but some cases have been described as having followed dog and cat bites—Tinel and Moncany (1929) and Thomas and Kudelski (1929). Such cases may, however, be due to infection by saliva; Pasteur himself thought that there was a special 'virus' in the saliva of the enraged dog.

The appearance of lymphœdema in the hands of workers with compressed-air machines has been described by Orbach (1934), and this observation may lend support to a neurogenic theory of the cause, since such workers are known to have been occasionally afflicted with the Raynaud phenomenon.

Nothing definite is known of the cause of post-traumatic œdema. The histopathology of specimens removed by biopsy shows nothing beyond dilated lymphatic spaces and a variable small-celled infiltration. The histology has been investigated by many observers, some of whom state that it is unlikely that evidence as to the cause will be found on these lines. M. zur Verth (1929) suggested a nervous origin, and thought that the condition was analogous to post-traumatic wasting of muscle and atrophy of bone. Braeucker (1931) thought the lesion was the result of a reflex due to local irritation, the second, i.e. efferent, half of the arc being the sympathetic fibres, and claimed to have cured the condition by ramisectomy. As will be shown later, our experience does not confirm this view.

All that one can say at present is that in these cases of post-traumatic œdema the normal process of repair following a contusion fails for some unknown reason, and that a disorder of the lymphatic mechanism is initiated at the site of injury and may spread up and down the limb. Whether this disorder be an embarrassed return flow or an increased output is not clear, but our cases have shown after local injection of dye a considerable delay before excretion.

The clinical and operative appearances and findings in these post-traumatic cases are exactly the same as in those of the spontaneous group, and are described in the following section.

5. The Spontaneous Group.—It must be admitted that this group has no clear-cut diagnostic features. It contains, however, a large number of cases which differ from those in the other groups in that no definite cause is apparent.

The two following cases from our series are typical examples:—

Case 6.—Male, 23 years, student, was perfectly healthy until two years ago. He then noticed that towards the end of the day his left ankle was slightly puffy. He is an intelligent, observant man and states that he is not aware of any injury or other cause, and that after inquiry he finds that no such cases have been known to occur in his family.

Since onset the condition has steadily deteriorated, and he now possesses a lower extremity bolster-like throughout its whole extent. The right leg is entirely normal. The condition of this patient is illustrated in *Fig.* 555.

Case 7.—Male, 20 years, boot and shoe operative, began to notice a swelling of the left ankle eighteen months ago. He denies any injury, and has not heard of any member of his

family who has had a similar condition. The trouble has within eighteen months involved the whole lower extremity and has extended a short way above Poupart's ligament. The right leg is quite unaffected.

Cases such as these occur in both sexes—our series shows a ratio of two female to one male. The condition appears to arise at all ages, and the estimated age of onset in our series ranges from as low as 6 years to as high as 58 years, but the decade 20 to 30 years contains the largest number of cases.

The condition develops insidiously, the first sign being usually a slight puffy swelling about the ankle. This slowly spreads towards the trunk, and may eventually involve the skin of the abdomen and flank. The progress of the disease is slow but relentless. It is no doubt in existence in an early stage for some distance beyond its apparent clinical limits. This is shown by the fact that it has appeared in the stump shortly after amputations which have been done for lymphœdema.

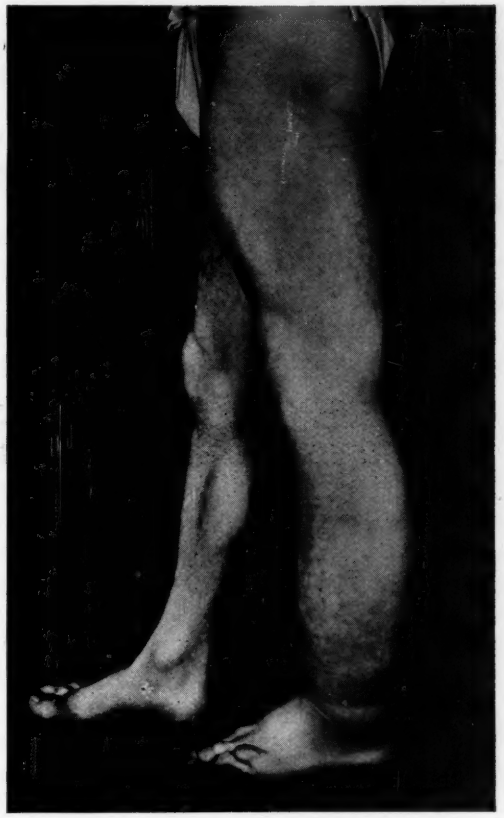

FIG. 555.—*Case* 6. Lymphœdema of the spontaneous type occurring in a man aged 23.

The limb may after some years reach an enormous size, but it remains of normal colour and is not liable to the chronic ulceration which is found so frequently in the 'white leg'. The disease is more commonly unilateral, but in some cases the opposite leg is eventually involved.

When an incision is made into the limb, as for a Kondoléon operation, the appearances are striking. The skin is much thickened; the subcutaneous fatty tissue is enormously increased in depth, and changes its character as one approaches the deep fascia. At first it is clear, almost translucent, and weeps quantities of clear lymph. Deeper down the layer is denser, with turbid lymph containing small flakes, whilst when the deep fascia itself is reached its outer surface cannot be plainly defined because it is overlaid with organizing lymph of a curdy appearance. The deep fascia is thickened, sometimes grossly, but when incised its deep surface is entirely normal. The disease never penetrates below the deep fascia. Portions of excised fascia show nothing more than an increase of well-formed fibrous tissue.

The cause of this fascial thickening is not clear; it has been thought by some that the thickening is the primary lesion, and that the disease results from obliteration of any communication between the superficial and deep lymphatics. But it has been known since Sappey that no such communication normally exists. From

the appearance of the outer surface of the deep fascia it seems clear that the thickening is not a primary lesion, but is due to the growth of young fibrous tissue by organization of the exudate.

Thickened bands along the external iliac vessels have been described and claimed as evidence of lymph stasis. In the course of five lumbar cord ganglionectomies which we have done in patients suffering from lymphœdema we have taken the opportunity to search upwards from the femoral canal very thoroughly, but entirely without finding any evidence of thickening either of lymphatic leashes or of glands. In this respect we corroborate the opinion of Basset, Haguenau, and Gauthron (1930), who exposed the iliac vessels in the course of a periarterial sympathectomy done for a lymphœdema of two years' standing in a woman of twenty-nine. These authors report no unusual finding, and state that the operation was without benefit.

Siccard, Haguenau, and Wallich (1928), in reporting a case of lymphœdema in a patient who suffered from spina bifida, quote Leri as having stated that the association of chronic lymphœdema with spina bifida is not uncommon.

Some German writers use the term "neurotisches Acro-ödem", and incriminate the autonomic system in various and often divergent ways, unbacked by experimental or clinical proof.

In spite of much work the aetiology of this type of chronic lymphœdema is still unknown.

TREATMENT

It is at the outset evident that methods such as rest, massage, and elastic pressure give purely palliative and fugitive results. Surgeons have attempted in the past to create new channels by the introduction of various foreign bodies, e.g., silk threads, and have ingeniously sought to drain the lymph via the femoral canal into the peritoneal cavity. Such methods have been rewarded by an immediate and dramatic success; but the good fortune has been short-lived, since the inevitable reaction of the tissues to a foreign body has always quickly choked the new channels.

Gillies and Fraser (1935) have reported an attempt to bridge the gap between diseased and healthy tissue in the lower extremity by the use of a flap of skin borrowed from the arm. Such a flap must be large, and there is risk, even in skilled hands, that the function of the donor arm might be seriously impaired.

Surgery to-day may be said to approach the attack on lymphœdema by one or other of two routes: (1) The attempt to utilize the normal lymph paths beneath the deep fascia (Kondoléon); (2) Some form of sympathectomy.

1. The Kondoléon Operation.—Kondoléon's first paper appeared in 1912. In this he described the operation of cutting long fascial strips, one end of each strip being left attached whilst the opposite end was thrust deep into the intermuscular spaces. His method is based on the work of Lanz, who in 1906 drilled the femur of a case and inserted the strips of fascia into the medulla. Kondoléon published a further paper in 1912, and gave an account of 7 patients on whom he had done the operation.

Hill (1915) suggested that after removal of the fascial strips the edges of the opening in the fascia should be stitched to the underlying muscle.

An adequate Kondoléon operation will involve a long incision stretching on the outer side of the limb from the trochanter to the external malleolus, and on

the inner side from the level of the saphenous opening to the internal malleolus. These incisions may be interrupted for 3 in. at the level of the knee to obviate any possible stiffness due to scar formation, and the inner incision will of course be planned to avoid the internal saphenous nerve and vein. We have found it best to carry the incision at once through the deep fascia. By everting the edges of the incision in the fascia it is possible easily to cut a wide strip of fascia and overlying tissue from each side of the incision. The wound is closed with particular care to hæmostasis ; no drain is necessary and the bandage should be firmly applied.

Since 1930 we have done six of these operations, all in the lower limb, and have recently been able to examine the result in five of these patients. In only one case is the result satisfactory. This is a woman of 46 years, who at the age of 33 fell and 'sprained' her right ankle-joint. The swelling persisted, and when we saw her, six years after the injury, it had reached the level of the knee and had disabled her for her work. The operation was in 1930, and to-day the leg shows a very trivial swelling easily controlled by bandage, and she cycles daily to and from her work in comfort.

The results in the other four cases can only be described as incomplete and disappointing. In no patient is the limb as big as before operation, but the improvement is slight, and in no case is it more than 25 per cent. It is, however, of interest to note that in three of the patients, in which six, five, and four years respectively have passed since the operation, any further upward progress of the disease has been arrested.

2. Sympathectomy.—The disappearance of the supra-malleolar cuff of œdematous subcutaneous tissue in erythrocyanosis following the operation of lumbar sympathectomy encouraged us to hope that a similar result might be obtained in chronic lymphœdema. Since August, 1936, we have submitted 5 cases of chronic lymphœdema of the lower extremity to sympathectomy.

Brief details of these cases are as follows :—

Case No.	Sex	Age	Type	Site
1	F.	12	Spontaneous	Right lower extremity
2	M.	22	Spontaneous	Left lower extremity
3	F.	19	Post-traumatic	Right foot and ankle
4	M.	58	Spontaneous	Right lower extremity
5	M.	23	Post-traumatic	Left lower extremity

The operation performed was that of lumbar cord ganglionectomy involving removal of the chain opposite the bodies of L. 2, 3, 4. This operation is mainly preganglionic for the lower extremity, and is known, from its results in vascular disorders, to be the most efficient type of sympathectomy for the lower extremity. This is more particularly true if the fourth lumbar ganglion is left intact.

The end-results of the operation in lymphœdema have been most disappointing. Whilst it is true that during the three weeks following the operation there occurs

a remarkable shrinking of the limb, much greater than is ever produced by rest alone, once walking and work are resumed the limb quickly reverts to its pre-operative size and shape.

We are regretfully driven to the conclusion that lumbar cord ganglionectomy (and by inference any lesser form of sympathectomy) has no place in the treatment of chronic lymphœdema of the extremities.

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DISLOCATIONS AND FRACTURE-DISLOCATIONS OF THE PELVIS*

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TYPES OF PELVIC RING INJURY

THE two innominate bones, by their articulation with the sacrum posteriorly and with each other at the symphysis pubis, form an intact pelvic ring. If a fracture of the pelvis breaks the continuity of this ring at only one level, displacement must be of minimal degree. On the other hand, if there is a secondary injury to another part of the ring, considerable displacement of the separated segment is possible.

Isolated Injuries of the Pelvic Ring.—The isolated injuries include fracture of the body of the ilium, unilateral fracture of one or both pubic rami, and slight

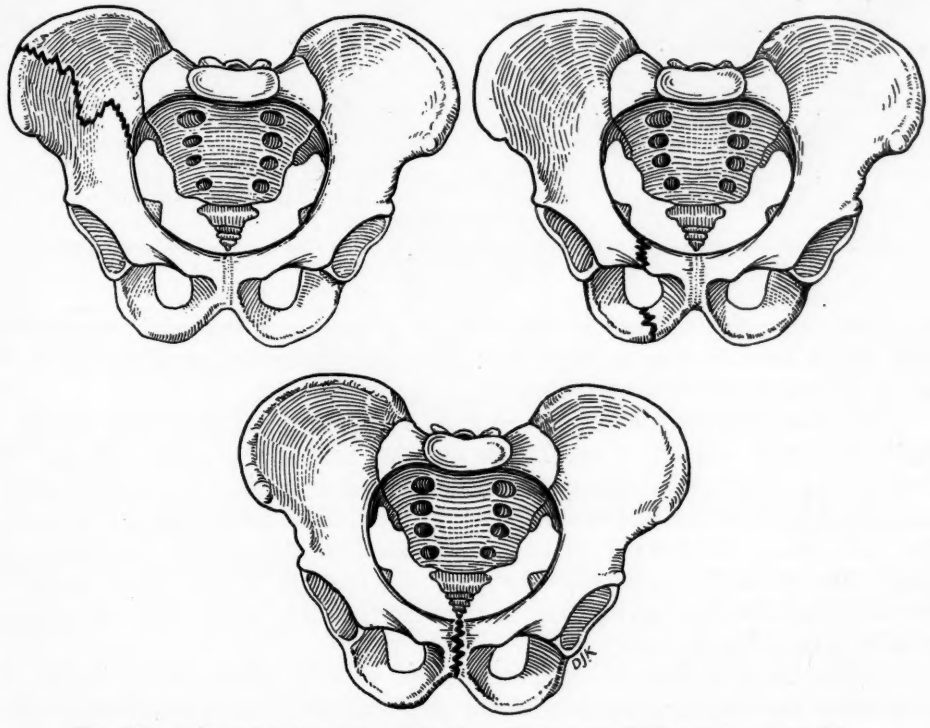

FIG. 556.—Isolated injuries of the pelvic ring. There is no displacement; no special treatment is necessary.

separations of the symphysis pubis (*Fig.* 556). Since there cannot be serious displacement, no treatment is required except recumbency for a few weeks. Immobilization is unnecessary, and the patient may be allowed to turn in bed and to lie in any position.

* Based on a demonstration to the Association of Surgeons, March, 1937, and part of a paper to the Canadian Medical Association, July, 1937.

Combined Injuries of Pubic Segment of the Pelvic Ring.—The combined injuries are of two types. The pelvic ring is made up of the anterior pubic segment, developed simply for the attachment of muscles, and of the postero-lateral iliac segments, which also serve the function of weight-bearing. The first type of combined injury is that in which both fractures lie in the anterior segment—bilateral fractures of both pubic rami, and unilateral fractures of the pubic rami with dislocation of the symphysis (*Fig.* 557). These injuries are produced by compression of the pelvis in its lateral axis; for example, the patient who is standing sideways to a wall is crushed by a motor vehicle which strikes the opposite side of the pelvis. The detached fragment of bone is relatively small. The displacement is limited in degree by the attachments of muscles, and it cannot alter the alinement of any weight-bearing joints or produce any shortening of the limbs.

FIG. 557.—Combined injuries of the pubic segment of the pelvic ring, produced by lateral compression. There is moderate displacement; patients must lie on their backs.

As a rule, therefore, it may be ignored. The patient is treated in recumbency, lying on his back for four to six weeks: the lateral compression of the pelvis must not be increased by allowing the patient to lie on one side.

Combined Injuries of Iliac and Pubic Segments of the Pelvic Ring.—In the second type one injury breaks the anterior half of the pelvis and the other involves the weight-bearing segment. The common examples are dislocation of the symphysis with fracture of the ilium, dislocation of the symphysis with sacro-iliac dislocation, and fracture of both pubic rami with sacro-iliac dislocation (*Fig.* 558). The pelvis is completely disrupted and one-half of the pelvic girdle may be widely displaced, carrying with it the corresponding lower limb, so that there is deformity and shortening.

Unlike the first type of combined injury, which is produced by lateral compression of the pelvis, these injuries are produced by forcible compression in the antero-posterior axis. The patient is standing with his back to the wall when he is crushed by a motor vehicle, or he is lying on the roadway and the wheel of the vehicle mounts one side of the pelvis. In three personal cases head-on collisions were responsible. Two motor cyclists met at speed in the middle of a straight and otherwise unoccupied five-mile stretch of road, and one sustained a unilateral pelvic dislocation (*see Fig.* 562). Three men weighing 14, 16, and 17 st. respectively were wedged into a two-seater car travelling at 60 miles an hour when they struck a telegraph pole with such violence that the pole fractured at the top, near the

overhead wires. The two outside men were shot out sideways uninjured, like peas out of a pod, and the central passenger sustained a fracture of the ilium and dislocation of the symphysis with pelvic disruption.

For these injuries many types of treatment have been described, and reduction of the deformity has frequently been difficult. Extension has been applied to one

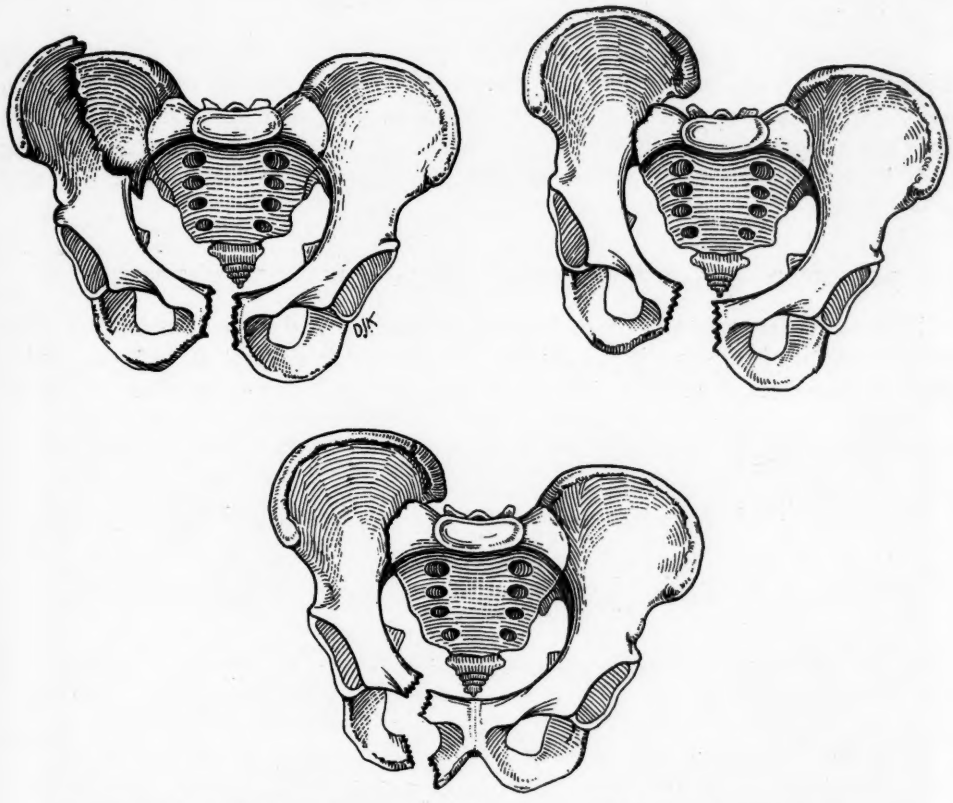

FIG. 558.—Combined injuries of the pubic and iliac segments of the pelvic ring produced by antero-posterior compression. There is severe displacement; patients must lie on their sides.

or both lower limbs, skeletal traction has been employed, pelvic slings and girdles have been devised, and the latest treatment includes a formidable operation involving exposure of both obturator foramina and the insertion of a 'grab-hook' with a screwing device to force the two halves of the pelvis together. In all of these procedures the patient has been treated in the recumbent position, lying on his back.

MECHANISM OF DISPLACEMENT IN DISLOCATION OF THE PELVIS

The antero-posterior compression which is responsible for unilateral dislocations gives a clue to the mechanism of displacement, and this is confirmed by radiographs, which show extraordinarily wide separation of the symphysis pubis, with a displacement at the sacro-iliac joint, which often appears to be minimal and which may even be overlooked altogether. Clinically it may be possible to insert

a fist between the two pubic bones, and yet there is only a trace of undue prominence of the posterior superior iliac spine. This is because the displaced innominate bone is rotated round a longitudinal axis near the sacro-iliac joint. The dislocated half of the pelvis is swung outwards, and only secondarily in the more severe cases is it displaced upwards into the loin. The whole weight of the lower limb maintains the outward rotation, and whatever manœuvre is practised while the patient lies on his back, replacement is difficult.

The dislocated pelvis is like a partly opened oyster. Balance the oyster on the hinge at its posterior margin, and whatever traction is applied to the two shells, gravity keeps them apart. Lay the oyster on its side, and quite simply the two halves close together. So it is with the dislocated pelvis. With the patient lying on his back reduction must always be difficult. Lay the patient on one side and the two halves of the pelvis fall together.

METHOD OF REDUCTION AND AFTER-TREATMENT

If there is a complete dislocation of the symphysis and sacro-iliac joint without visceral complication, the dislocation is reduced under general anæsthesia, and a double plaster spica is applied. A plaster table or any form of pelvic rest may be

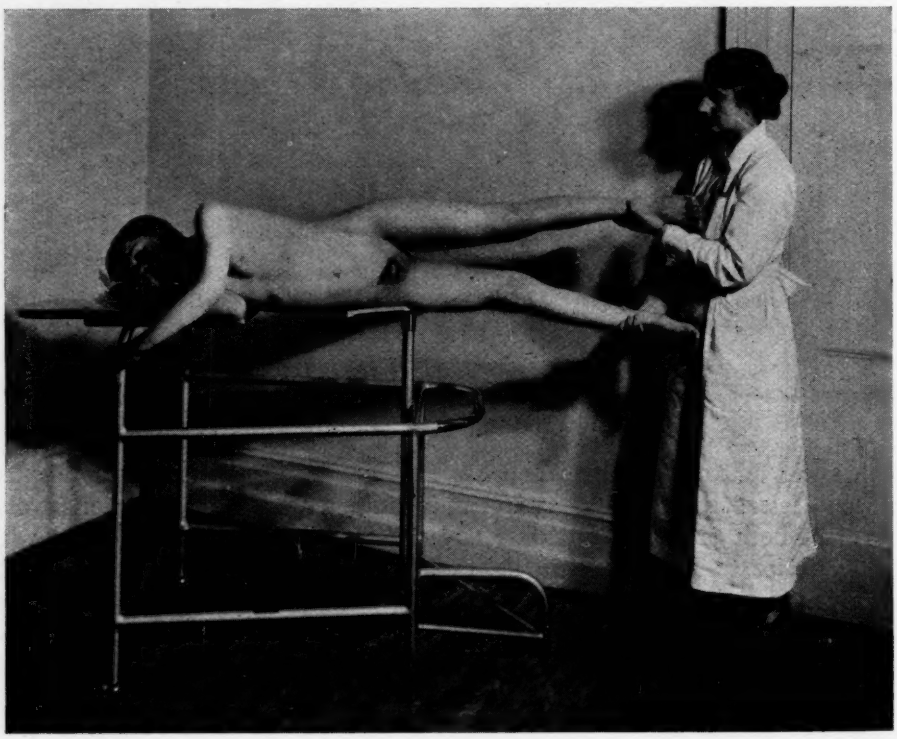

FIG. 559.—Reduction of dislocations and fracture-dislocations of the pelvis by lateral recumbency.

used with the perineal post removed. The patient is placed on his uninjured side, with the trochanter lying on the pelvic rest and the two lower limbs held one above the other by an assistant (*Fig.* 559). In many cases the dislocation is already reduced

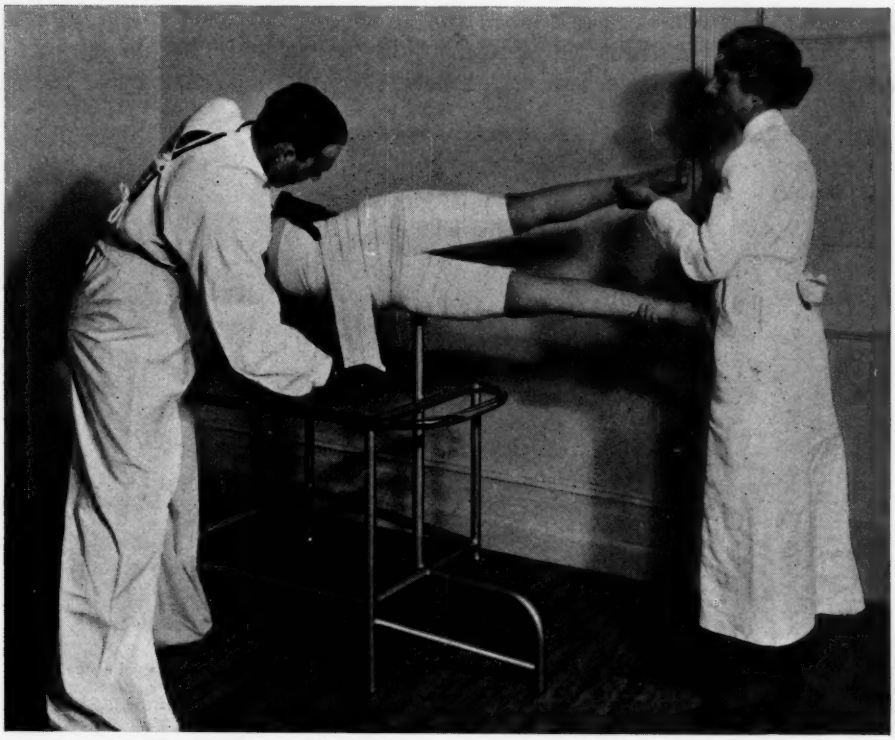

FIG. 560.—The dislocated ilium is rotated forwards and downwards towards the uninjured side.

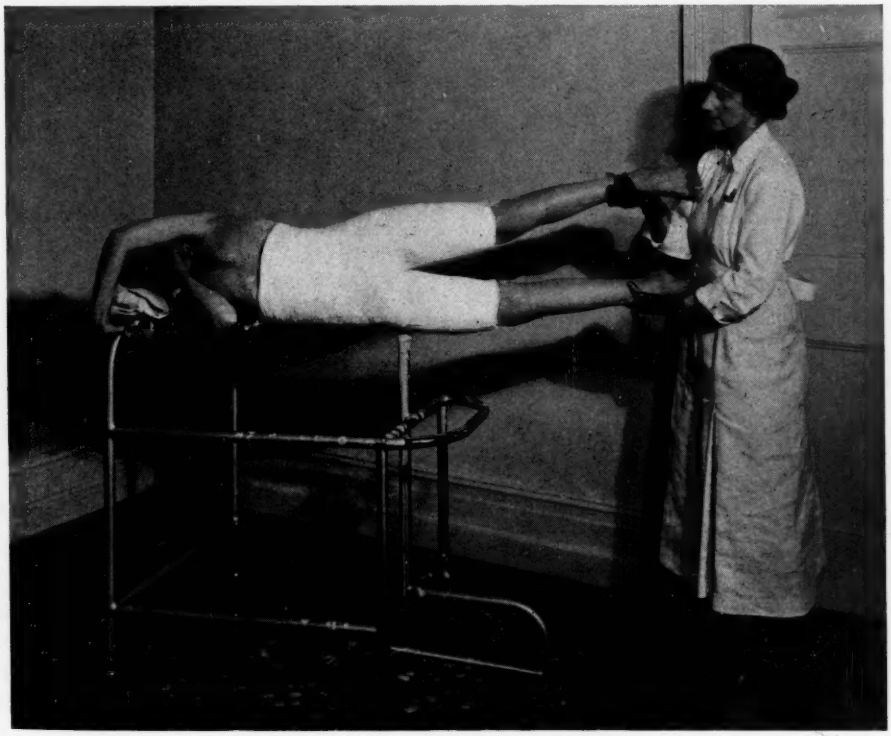

FIG. 561.—A double plaster spica is applied.

by the time the patient is in this position. It may be necessary to apply pressure over the crest of the dislocated ilium, pushing and rotating it downwards and forwards towards the normal half of the pelvis (*Fig.* 560). Accuracy of reduction

FIG. 562.—Complete unilateral dislocation. The sacro-iliac dislocation may easily be overlooked. Comparison with the normal side of the position of the pubis in relation to the midline, the shape of the obturator foramen, the prominence of the ischial spine, and the overlap of the back of the ilium over the sacrum, proves the typical rotatory displacement of the left innominate bone.

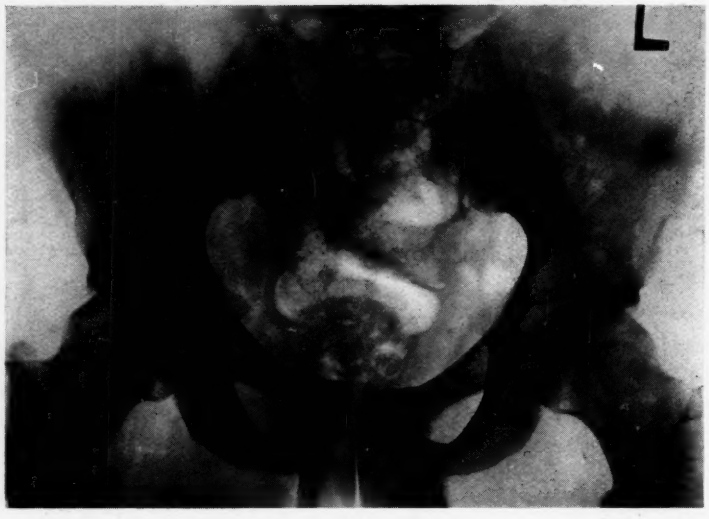

FIG. 563.—Same case as *Fig.* 562, twelve months after reduction by simple lateral recumbency and double plaster spica. Perfect functional recovery.

can be confirmed by palpating the pubic bones through the lower abdominal wall and in the perineum. If there is any doubt, a radiograph is taken before the plaster is applied.

The iliac crests are protected with adhesive felt and a double plaster spica is applied (*Fig.* 561). The spica must be closely moulded to the pelvis and lumbar

region, and the minimum of wool padding is employed. The pelvic rest is cut out, the defect in the plaster repaired, and a post-reduction X-ray is taken through the plaster. Throughout the period of recumbency the patient should be encouraged

FIG. 564.—Old unreduced sacro-iliac dislocation with pubic fractures. The rotatory displacement is very obvious in the position of the head and neck of the femur on the dislocated side. There was permanent shortening and deformity. The sacro-iliac pain ultimately subsided because bony ankylosis supervened.

FIG. 565.—Unilateral dislocation with minimal displacement (sacro-iliac subluxation with two chip fractures of the ala of the sacrum, and symphysis subluxation with bilateral pubic fractures).

to lie on one side and not on his back. If the plaster becomes loose in two or three weeks' time a new spica is applied, again in lateral recumbency. Immobilization is continued for three months, and periodic check radiographs are advisable throughout. (*Figs.* 562, 563.)

The same routine may be adopted when, instead of a dislocation of both joints, there is a dislocation of one joint and a fracture near the other. In these cases, however, there is greater mobility of the displaced segment of the pelvis, reduction is less stable, and it is easy to over-reduce so that the dislocated pubis lies behind the body of the pubis on the normal side. If there is any tendency to recurrence of upward and outward displacement within the plaster, skin traction may also be used on the injured side, but as a rule recurrent displacement indicates looseness of the plaster. In fracture-dislocations of the pelvis where the sacro-iliac joint is not injured, absolute accuracy of reduction is less essential than in the injuries where the sacro-iliac joint is dislocated. Even considerable displacement of the symphysis pubis may exist without any functional disability. On the other hand,

FIG. 566.—Same case as *Fig.* 565. No treatment except nursing in lateral recumbency; the patient was not allowed to lie on his back for six weeks.

unreduced sacro-iliac dislocations, even of minor degree, account for persistent backache unless the injury has been so severe that the joint undergoes bony ankylosis (*Fig.* 564).

With minor separations of the symphysis pubis, especially when there has been no sacro-iliac subluxation, it may only be necessary to nurse the patient on his side on a firm mattress with fracture boards (*Figs.* 565, 566). Furthermore, when the pelvic disruption is complicated by rupture of the urethra, extravasation of urine, or injury to the pelvic viscera which interferes with active treatment of the bone and joint injury, simple lateral recumbency should be adopted.

ANALYSIS OF CASES

Records are available in hospital and private practice of 18 cases of total disruption of the pelvis—the second type of combined injury of the pelvic ring. These cases are from a total series of 143 pelvic fractures and dislocations (excluding marginal fractures of the acetabulum, central dislocations of the hip, and injuries of the sacrum and coccyx). The incidence of total disruption is probably not so

high as these figures suggest, because a large number of minor pelvic fractures may not be referred to orthopædic surgeons.

The commonest type of total disruption was unilateral dislocation without fracture.

Dislocation of symphysis with dislocation of sacro-iliac joint ..	9
Dislocation of symphysis with fracture-dislocation of sacro-iliac joint	2
Dislocation of symphysis with fracture of ilium	4
Fracture of pubic rami with dislocation of sacro-iliac joint ..	3

Six of the cases were old unreduced injuries when first seen (*Fig.* 564). One of these was the youngest patient in the series—a child of $3\frac{1}{2}$, who sustained a complete unilateral dislocation. The striking feature of these cases was the constant complaint of aching pain in the sacro-iliac region when this joint was involved, and the absence of symptoms other than shortening of the leg when the joint was uninjured.

One patient had sustained a rupture of the urethra with extravasation of urine, and no active treatment was possible other than simple lateral recumbency. The other eleven cases were reduced by manipulation and plaster in lateral recumbency. One, a child of 9, died three days after a severe crushing injury. All the other patients made uneventful recoveries.

SUMMARY

1. Fractures of the pelvis may be subdivided into injuries not involving the pelvic ring (fractures of the crest and spines of the ilium, fractures of the acetabulum, and fractures of the sacrum and coccyx), and fractures involving the pelvic ring.

2. Isolated fractures of the pelvic ring cannot be widely displaced. They are treated by simple recumbency; the patient may lie in any position.

3. Combined fractures of the pubic segment of the pelvic ring are produced by lateral compression of the pelvis. There cannot be severe displacement. They are treated by simple recumbency; the patient must lie on his back and not on one side.

4. Combined fractures of the iliac and pubic segments of the pelvis cause complete disruption and wide displacement. They are produced by antero-posterior compression of the pelvis. The displacement is most easily corrected by manipulation in *lateral* recumbency and a double plaster spica. The patient must lie on one side and not on his back. The functional result depends on the accuracy of reduction of the sacro-iliac dislocation, which may easily be overlooked.

THE PATHOLOGY OF THE SINGLE NODULE OF THE THYROID GLAND

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Some explanation may appear to be necessary for re-opening a discussion on the pathology of the single nodule in the thyroid gland.

Although there is general agreement about the nature of multiple nodules, the single nodule is still a matter of controversy. Formerly the multiple nodules characteristic of the nodular or 'adenomatous' goitre were considered to be neoplasms. All the evidence, however, goes to prove that these nodules are produced by localized areas of hyperplasia followed by involution. It is now known that all colloid goitres tend to become nodular with age. It is probably true to say that all simple goitres in people over the age of 25 will show evidence of nodularity in the cut surface, although clinically the swelling may have been smooth and uniform.

The nature of the single nodule, however, occurring in the otherwise normal gland continues to give rise to speculation. Even the most recent writings evade the question. I quote from J. H. Mean's[1] excellent work on the thyroid published recently: "Our own belief is that so-called colloid adenomata are not tumours and that so-called fœtal adenomata, or at least some of them, are. Even the pathology, however, is very confusing. One finds a veritable spectrum of types merging one into the other, from a nodule made up of perfectly innocent looking normal, or colloid, tissue, through ones which look like fœtal tissue, to undifferentiated types with blood-vessel invasion, which are clearly malignant. These one hears spoken of sometimes as malignant adenomata."

Sir Thomas Dunhill[2] writes: "Nodular involution goitre may occur as a single nodule in a normal or comparatively normal thyroid, the common so-called 'colloid adenoma' or cystic adenoma. . . . In the case of the single nodule the gland as a whole has met the physiological emergency normally with the exception of one lobule. In some cases the formation of a colloid nodule is due to colloid degeneration taking place in a true adenoma."

It is thus seen that there is no uniformity of opinion about the nature of these single nodules, although all authorities hint at the existence of a true innocent neoplasm. The present investigation has been undertaken in order to decide whether these single nodules are neoplasms or localized areas of involution.

ANALYSIS OF CASES

Material.—This consisted of all cases on the Surgical Unit in which at operation the remainder of the gland appeared normal to the naked eye and no other nodules could be felt. When the condition of the rest of the gland had not been noted the case was excluded from the series. This does not assume that in cases of multiple nodules the nodules are all of the same nature; but it was

considered good policy to limit the investigation strictly to cases in which only one nodule had been found.

Number of Cases: 88.

Frequency in this Series: Out of 943 cases of goitre there were 88 cases in which the enlargement of the gland was a localized nodule.

Clinical Evidence in Favour of Neoplasm.—It is reasonable to suppose that an involutionary process would be generalized in distribution and would continue to occur for an indefinitely prolonged period of time; so that if a nodule resulting from involution were removed, a further nodule might be expected to develop. This is in fact what occurs in cases of multiple nodules. In order to decide whether the single nodule behaves similarly the cases were followed up.

Follow-up: Cases between 1927 and 1934 were followed up. As most of the patients were young unmarried women who changed their names at least once, and their addresses frequently, it was found impossible to go further back than 1927. Cases since 1934 were considered too recent. Of the 33 cases written to, 27 replied. In none of the cases traced had further nodules developed.

Aetiological Data.—

Age Incidence: From 14 to 68 years, average age 32.

Under 20	.. 5 cases	30–40	.. 26 cases
20–30	.. 31 „	Over 40	.. 26 „

Length of History: From 6 months to 25 years, average 6 years.

Average Age of Appearance: 26 years.

Sex: Males 12, females 76.

Size: ½ to 3½ in. in diameter, average 1½ in.

Position: About half were in the right lobe; the remainder were divided equally between the left lobe and the isthmus.

Symptoms.—In 12 cases out of 88 there were symptoms. Eight patients complained of slight dyspnœa on exertion and occasional palpitations. In the remaining four cases there was some complaint of pressure symptoms; in one of these the symptoms came on suddenly, and recent hæmorrhage was found on examination of the specimen.

It has been argued that, whatever the nature of the single nodule may be, the matter is not of practical importance, as this condition cannot be distinguished clinically from multiple nodular goitre. This is not true in the majority of cases, for although the trachea may be distorted by a more or less uniform enlargement of the thyroid (as in multiple nodular goitre), it is never so grossly displaced laterally as it frequently is by a single nodule (*Fig.* 567). Further, the clinician is unable, as a rule, to feel the remainder of the gland when a single nodule is present, whereas there is palpable enlargement of the whole of the thyroid in cases of multiple nodular goitre. Unless the lateral displacement of the trachea is borne in mind, it may be mistaken for enlargement of the second lobe. Hertzler,[3] moreover, points out that the single nodule tends to extrude itself from the gland, and therefore forms a more prominent swelling than one of the nodules of a multiple nodular goitre.

Pathology.—Twenty out of the 88 solitary nodules have been described in the case notes as fœtal adenomata. The fœtal adenoma is a tumour with characteristic macroscopic and microscopic features.

Macroscopic.—The well-marked capsule stands out as a characteristic feature. The striking appearance of this envelope contrasts with the thin and more irregular capsule of the involution nodule. The contrast is well seen in a specimen of a

FIG. 567.—Section through thyroid and surrounding structures, showing adenoma in lateral lobe displacing trachea. Found post mortem. *(Kindly lent by Mr. Geoffrey Keynes.)*

'fœtal' adenoma occurring in a nodular goitre (*Fig.* 568). The adenoma can be shelled out like a fibro-adenoma of the breast, in strong contrast to involution nodules. The tissue itself is typically uniform in consistency, homogeneous, and greyish-yellow in colour. Later in the life-history a fibrous core develops which

FIG. 568.—Adenoma in a nodular goitre, showing well-marked capsule.

is typically central but often eccentric, and from which fibrous septa radiate, resembling a grape-fruit. Still later, when degenerative changes take place, hæmorrhage, liquefaction, and calcification occur.

Histology.—There are three distinct features characteristic of fœtal adenomata: (1) Capsule; (2) Acinal cells; (3) Fibrous septa.

1. *The capsule*: This is thick and regular, differing considerably from the thinner and more irregularly arranged structure of the capsule surrounding the nodules of a nodular goitre.

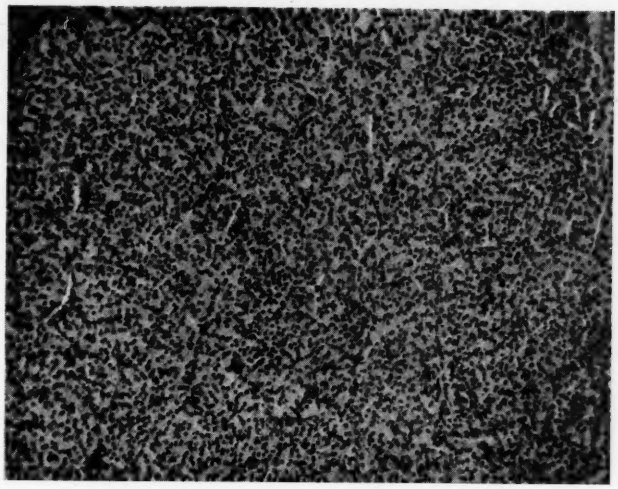

FIG. 569.—Adenoma showing cells arranged in columns without acinar formation. (× 56.)

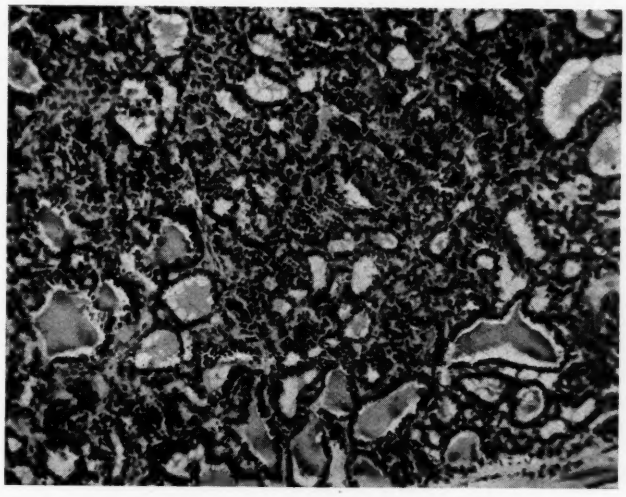

FIG. 570.—Adenoma with fully formed vesicles containing colloid. (× 56.)

2. *Acinal cells*: The acinal cells are polyhedral and contain large dark-staining nuclei and very little faintly-staining protoplasm. Early in their life-history they lie in sheets without lumina or are arranged in columns. The lumen gradually forms and a minute amount of colloid appears, the structure now resembling that of the thyroid gland in a newborn child. Development proceeds and large vesicles filled with colloid appear, but the cells lining them remain small, with large dark-staining nuclei. At this stage distinction between a nodular goitre and a fœtal

adenoma would be difficult from a study of the acini alone were it not for the constant presence of areas of less well-differentiated tissue. The adenoma may be composed of undifferentiated cells only, or more often all the stages may be seen in one section or even in one microscopic field. (*Figs.* 569, 570.)

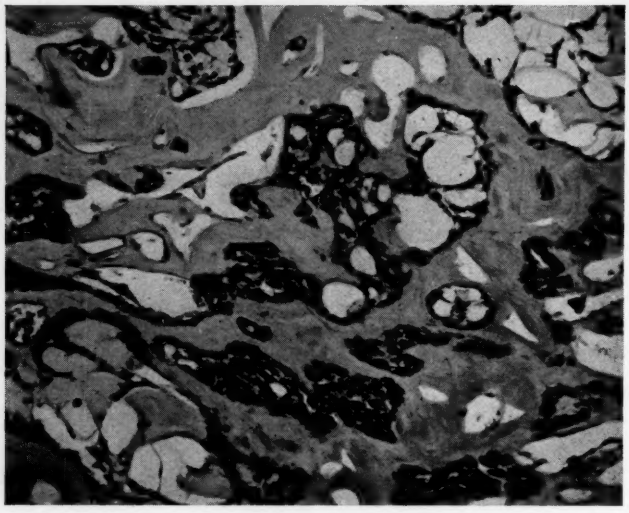

FIG. 571.—Section of adenoma showing fibrous septa swollen and pale—early degenerative change. (× 56.)

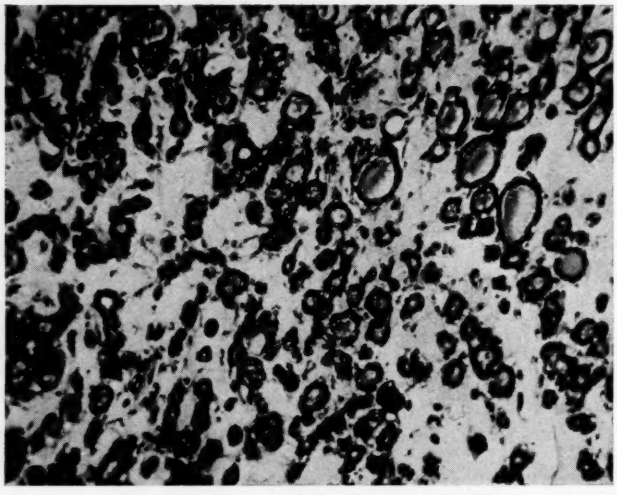

FIG. 572.—Adenoma showing liquefaction of fibrous stroma. (× 56.)

3. *Fibrous septa*: Well-marked fibrous septa are characteristic, and play an important part in the changing histological appearances. Degeneration occurs first in the septa, which become swollen and pale, disintegrating to form structureless non-staining masses. The acini become separated and lie loose in the gelatinous stroma, finally disappearing and leaving a cyst. The degeneration of the fibrous tissue walls, consequent lack of support, and probably degenerative changes in the blood-vessels, lead to hæmorrhage. (*Figs.* 571, 572.)

Examination of the remaining 68 nodules shows in every case areas of relatively undifferentiated tissue, usually lying just beneath the capsule, but often interspersed between vesicles which resemble more closely the adult type.

Whether the tumour has been classified as a fœtal adenoma has depended upon the quantity of undifferentiated epithelial tissue present. A histological analysis of these specimens shows, however, that there is no essential difference between them. The variations in appearance under the microscope depend entirely upon the degree of differentiation and the extent of degeneration in the tumour.

DISCUSSION

Before discussing critically the possible origin of these solitary nodules it is necessary to define clearly the points at issue: (1) Is the solitary nodule the result of a localized process of irregular hyperplasia and involution or is it a neoplasm? (2) If the nodule is a neoplasm, from what tissue does it arise?

1. It is well established in large series of cases derived from districts where goitre is endemic that the goitres which were diffuse and uniformly enlarged in early life become nodular as age advances (Wegelin, quoted by Graham[4]). These multiple encapsulated nodules were originally thought to be neoplastic, this view meeting with general acceptance for many years.

Even now the point cannot be considered to be finally settled. However, there is strong evidence in favour of irregular hyperplasia and involution to explain the origin of the multiple nodular goitre, and the terms 'multiple adenomata' and 'adenomatosis' must be regarded as misnomers. This paper is not concerned with the origin of nodular goitres, except in so far as it may be of assistance in elucidating the origin of the single nodule. The evidence in favour of hyperplasia and involution may be briefly summarized as follows: (*a*) In endemic areas diffusely enlarged glands tend to become nodular with advancing age, and in direct relation to the intensity of the endemic influences. (*b*) The multiplicity of the nodules. (*c*) Histological evidence—all the stages in the evolution of the nodule can be traced from the unencapsulated localized area of hyperplasia up to the isolated encapsulated involutionary nodule.

The important points to stress in reference to the present question is that the process begins in an abnormal gland and that the multiplicity of the nodules throughout the gland suggests the 'diffuseness' of the process.

That the single nodule may arise in a different way is suggested by the following facts: (*a*) It occurs in younger people. The large majority of single nodules are first noticed between 20 and 30 years of age, whereas the maximum incidence of nodular goitre is between 30 and 50. (*b*) The remainder of the thyroid gland is macroscopically normal. (*c*) Excision of the solitary nodule is not followed by the formation of further nodules.

These facts surely suggest very strongly that the solitary nodule differs in origin from the nodular goitre, and is probably different in its nature also—that it is, in fact, an innocent neoplasm.

2. Passing now to the second question—If the solitary nodule is a neoplasm, from what tissue does it arise?—there are three possibilities: (*a*) That the tumours may arise from embryonic rests—'fœtal' adenomata. (*b*) That they may arise from adult tissue. (*c*) That there may be two benign neoplasms—the 'fœtal' adenoma and the 'adult' adenoma.

a. Whether or not Wolfler's views on the origin of multiple nodules in an adenomatous goitre from fœtal cell-rests are to be believed, the lesion described by him as fœtal adenoma has been recognized ever since as a pathological entity. Wolfler postulates that portions of the original anlage of the thyroid remain as embryonic islands, isolated by connective-tissue capsules, and lying dormant or failing to respond to the stimulus of growth until a subsequent period of life. This theory, if critically examined, possesses several defects. The thyroid is one of the first organs to be differentiated in the human body and can be recognized in the 1·5 mm. embryo (third week) (Norris[5]). Follicles are formed at the 24-mm. stage (eighth week), and thereafter development proceeds apace. Lucas Keene and Hewer[6] have shown the presence of colloid in a fœtus of 11 weeks, and conclude that at full term the thyroid is actively secreting. If, therefore, embryonal rests occur, they must be isolated incredibly early in embryonic life, because they consist of undifferentiated cells in the prefollicular stage. Investigators have had great difficulty in finding embryonic rests in normal thyroids of embryos, infants, or children. If they occur, their incidence is out of all proportion to the incidence of fœtal adenomata.

b. The possibility of adenoma arising from adult tissue must now be examined. In between groups of acini there are collections of epithelial cells, the interacinar parenchyma. The origin and even the very existence of these cells seems to be a matter of controversy. Rienhoff[7] considers that they do not exist and that the appearance might be due to follicular epithelium cut tangentially. The cells, however, differ from the adult epithelium, and resemble closely the undifferentiated cells. They are not encapsulated, and are therefore distinct from embryonic rests; moreover, they do not show the characteristic lobulation of fœtal tissue. It seems more likely that they bud off from the adult follicle and are capable of forming fresh vesicles. They are more numerous at birth, and steadily diminish in number in the adult gland. Whether or not the interacinar cells form new vesicles, it is quite certain that in hyperplastic states the thyroid epithelium is capable of dividing and producing immature cells.

If it is accepted that the follicular epithelium actively divides and forms fresh vesicles, it is not difficult to believe that under the unknown stimulus to neoplasia adenomata may form from adult tissue.

c. The third possibility, that there are two benign neoplasms of the thyroid, the 'fœtal' and the 'adult' adenoma, must be discussed. The histological analysis of this material not only shows all gradations from thyroid tissue resembling that of early embryonic life up to tissue resembling that of the adult gland, but can be carried further, through stages of degeneration seen in abnormal involution in the pathologically enlarged thyroid gland. In some tumours the quantity of undifferentiated tissue is greater than in others, but in each case every gradation of differentiation can be found. These facts point to a common origin. Either they are all fœtal adenomata arising from dormant cell-rests which gradually grow under some abnormal stimulus and differentiate to a greater or lesser degree, or they are all formed by dedifferentiation from adult follicles. We maintain that the evidence obtained from examination of these 88 nodules substantiates the latter view. The undoubted rarity of embryonic rests and the frequency of the tumours (roughly 10 per cent of all goitres in our series), and the fact that a cellular very 'fœtal' tumour may be seen in patients over 50, both point to the view that the tumour arises from adult epithelium.

SUMMARY

Eighty-eight solitary nodules in the thyroid gland have been critically examined from both clinical and pathological points of view in order to decide whether they are benign neoplasms, and if so what their life-history may be. The evidence suggests that these nodules are innocent neoplasms, and that they arise from adult tissue in the same way as a fibro-adenoma forms in the breast. They vary considerably in their degree of differentiation, and range from a very cellular tumour composed of undifferentiated cells up to tumours at the other end of the scale which have the characters of the adult thyroid gland. They show a general tendency to develop into the adult type of thyroid tissue as they grow, and are prone to early degeneration leading to the formation of cysts.

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FIFTY-THREE CASES OF VESICAL DIVERTICULA

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In 1925 I published in the *British Journal of Surgery* "A Clinical Study of Eleven Cases of Vesical Diverticula". Since that date I have seen a good many more examples of this condition, and now report upon a total of 53, which figure includes the first 11 cases. Case summaries of the earlier series are here presented together with those of the more recent patients, as they were not given in the original article. Many of the cases are discussed in some detail, and are used to illustrate the methods of diagnosis and treatment, and the complications which occur in this disease. In all but 4 instances, *Cases* 15, 26, 43, 51, some form of operative treatment was undertaken, diverticulectomy being performed in 36 cases.

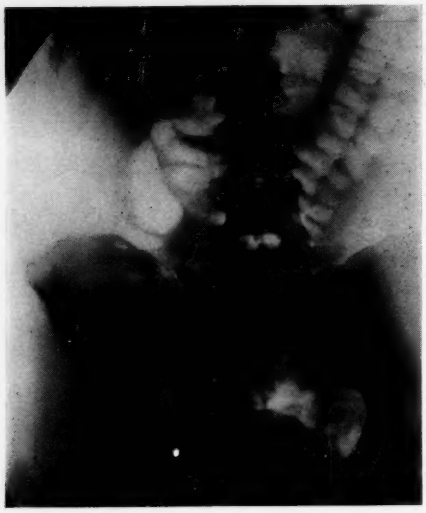


FIG. 573.—*Case* 33. Cystogram. The bladder outline is irregular; the diverticulum is smooth and lies above it.

In this series of 53 cases the average age is 57; the youngest was 15, the next 18, and the next 32; there were 10 in the forties; and the oldest was 80. All were males except *Case* 51, a female suffering from urethral stricture, whose diverticulum is not yet large enough to require resection.

In only 2 cases can the diverticulum be considered of congenital origin; one was the boy aged 15 years. The history of this patient was given in full in the publication already referred to, and is again summarized here (*Case* 7). The other young patient was aged 18 (*Case* 33). Though he had suffered always from enuresis and from frequency of micturition by day, it was not until this age that there arose any suspicion of bladder disease. Then hæmaturia occurred, and lasted for three days. The urine became obviously purulent, and at first contained *B. coli* and later staphylococci and *B. proteus*, and there were many subsequent attacks of cystitis and right pyelitis. Six ounces of residual urine were found. Cystoscopy and a cystogram (*Fig.* 573) showed a pouch almost as large as the bladder, with its opening in the region of the right ureter, which, however, was found not to be involved when the diverticulum was resected. One and a half years after this, the first operation, right nephrectomy was performed, a very small infected hydronephrotic kidney with a hydro-ureter being removed. Such changes in the upper urinary tract not uncommonly result from the presence of a diverticulum, and are presumably due to its direct pressure upon the ureter, for both ureters are not necessarily

affected. Following the diverticulectomy the residual urine fell to 1½ oz., but after seven years it still remained at this figure and the infection persisted, but in much lessened degree. At this time a new cystogram (*Fig.* 574) showed that there was now a weakness in that part of the right side of the bladder wall from which the diverticulum had been resected, and the bladder was again explored. A further operation was undertaken, and a thin-walled pouch anterior to the right ureteric orifice was defined and excised, and the bladder wall repaired. The residuum of the right ureter was not itself dilated. The internal urinary meatus and posterior urethra were patulous down to the triangular ligament, and there was no evidence of stenosis of the col vesicæ. As, however, there was an overhang of

FIG. 574.—*Case* 33. Cystogram, showing irregular bulging at the site of diverticulectomy.

the middle portion of the prostate in this region, a wedge-shaped resection was performed, the trigonal mucosa being sutured down into the posterior urethra to form a well-marked gutter. About this time a neurologist investigated the case, but was unable to demonstrate the presence of any nerve lesion; however, it seems clear that a dysfunction of some sort existed at the bladder neck, and to this must be attributed the development of the diverticulum and its tendency to re-form after a lapse of time. Other possible explanations are that the intrinsic nerve-supply of the bladder was injured by the first operation, or that adhesions which formed between the bladder and the pelvic wall led to the recurrence of the condition. It may be that in some of these cases there is a disorder of the autonomic nerve-supply to that portion of the bladder in which the diverticulum develops, for the methods of testing the function of this system are necessarily very imperfect. *Case* 44 may

perhaps belong to this group, though vesical obstruction seems the more likely cause.

These pouches are, I believe, acquired conditions in all but the rarest instances, and they are very commonly associated with obstruction to the bladder outflow. It is true that many of them occur in those parts of the bladder where development is complicated by the entrance of the ureters, and where there may perhaps be an innate weakness, but it is unusual to find them before the patient has reached an age at which the existence of such obstruction may be expected, and in the great majority of cases this can be demonstrated to be at the vesical neck, though it is sometimes below it.

In this series of 53 cases there are only 2 examples of really huge diverticula (*Cases* 44, 46), but there are 24 which I have classified as large. Thirteen are of moderate size; in 6 patients the diverticula, though considered to require treatment, were smaller. Sacculation of the bladder is of course a condition very often met with, and associated with this there are sometimes one or more recesses worthy to be called diverticula, but the large solitary diverticulum in an otherwise normal bladder is an uncommon surgical abnormality. I have only 7 examples of these occurring in bladders which were free from sacculation and with little trabeculation, though in 6 other bladders of this character there was in each case one large diverticulum associated with smaller ones, which varied in number from one to three—that is to say, there were 13 cases in which the bladder showed no generalized signs of stress. A diverticulum is not often seen in an early stage, and it is rare when performing a routine cystoscopy to find a single recess, small in size, which one can predict will presently grow into a diverticulum of surgical importance. Indeed it is my experience that usually diverticula do not come under observation until they have attained considerable dimensions.

SYMPTOMS AND ASSOCIATED CONDITIONS

A vesical diverticulum does not give rise to any symptoms which are unique, and it is only rarely that its presence can be detected by general physical examination. The patient is commonly unaware that there is anything wrong with him unless his urine becomes infected, or until increasing difficulty or frequency of micturition arouses his interest.

Frequency of Micturition.—This is the commonest symptom, and is not due so much to the diverticulum itself as to associated cystitis or prostatic enlargement.

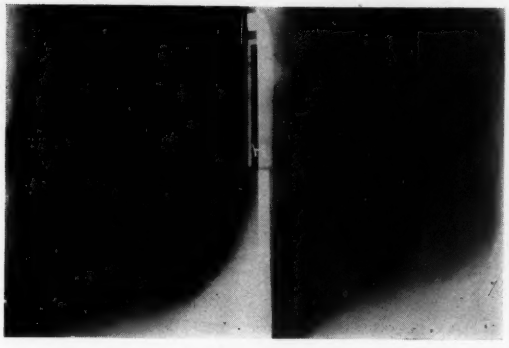
Pain on Micturition.—This is the next most usual symptom, and is also commonly due only to cystitis. In this series infection was present in all but 2 cases. Apart from discomfort of this sort, 2 patients with large diverticula had pain in the perineum, 2 had pain in the groin on the same side as the pouch, 4 had suprapubic pain, and 1 had pain along the sciatic nerve.

Hæmaturia.—In 18 cases there was a history of hæmaturia, and in 4 of these the diverticulum had a neoplasm in relation to it; in the others the bleeding was probably prostatic in origin or due to cystitis, but in some its source was obscure. In one instance, *Case* 38, there were papillomata in other parts of the bladder.

Difficulty of Micturition.—This is undoubtedly the most characteristic symptom. Thirty-six patients suffered from it, and out of this number 14 either

had retention of urine or had suffered from it intermittently. Such difficulty is not necessarily due to the presence of a diverticulum, for in 30 of these cases prostatic obstruction was also present. One patient found that he was free of difficulty in urinating provided that he did so whilst lying on his back. Only one patient gave the supposedly

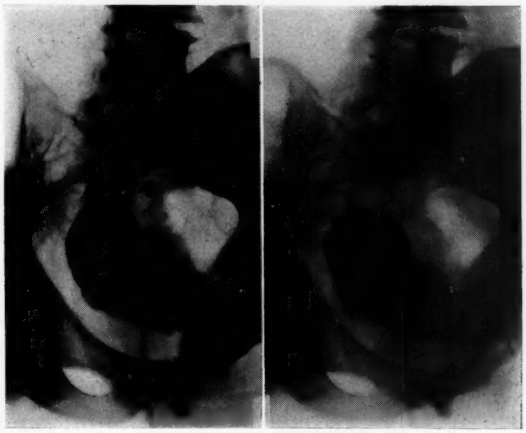
FIG. 575.—*Case* 30. Cystogram. The bladder is small and its outline irregular; the diverticulum, large and smooth, lies in the right side of the pelvis (left of illustration).



A B

FIG. 576.—*Case* 30. Cystograms (lateral radiographs). A, At rest; the bladder is anterior, the diverticulum posterior and larger. B, Straining to micturate; the bladder becomes very small, the diverticulum much enlarged.

diagnostic symptom of *miction en deux temps*. It is not easy to decide exactly how important a factor is the diverticulum itself in causing difficulty in the act of micturition. Most of these pouches have very thin walls, consisting of fibrous tissue and a few muscle fibres, and their contractile power is very slight relative to the rest of the bladder wall, so that when the patient begins to micturate and the fluid pressure rises, urine passes into the diverticulum at least as easily as into the urethra, and even more easily if there is an obstruction to the natural exit. This is well seen in the cystograms of *Case* 30. The antero-posterior view (*Fig.* 575) shows a large smooth-surfaced diverticulum on the right, and the irregular surface of the bladder on the left. It will be observed from the two lateral views (*Fig.* 576) that when the bladder contracts, the diverticulum, which is already much bigger than the bladder, enlarges still further. This patient had retention of urine, and the catheter had been withdrawn after being used to run in the opaque fluid. In the next, *Case* 24 (*Fig.* 577), a plugged catheter was in the urethra; the increased



A B

FIG. 577.—*Case* 24. Oblique cystograms. A, At rest; the diverticulum is seen above the bladder. B, Straining to micturate; the bladder diminishes, the diverticulum enlarges.

distension of the pouch is here not so marked, but is definite. These radiographs show also that there is no effective sphincter action at the orifice of the diverticulum.

The mechanism by which a diverticulum causes difficulty in micturition and perhaps retention has not been precisely determined. It has been suggested that such a pouch, as it fills, compresses the urethra from before backwards, but the lateral radiographs (*see Fig.* 576) do not support this view, as the diverticulum is seen to lie too high in the pelvis, though it is well known that diverticula often extend deeply in the direction of the pubic spine, for this state of affairs is commonly found at operation. An alternative idea is that, as the pouch fills, the urethra is so stretched that it cannot open normally. More probably it is a matter of fluid pressure; a rise of this within the bladder up to a certain level is necessary for relaxation of the internal sphincter, and the existence of a large and yielding

FIG. 578.—*Case* 26. Cystogram, showing concealed residual urine above, and a small amount in the bladder cavity below.

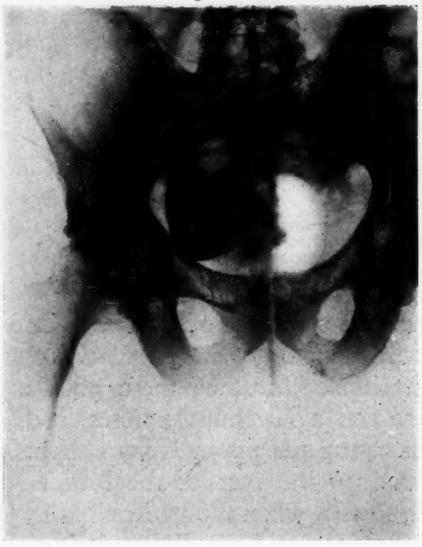


FIG. 579.—*Case* 30. Cystogram, showing concealed residual urine, the bladder having been emptied by catheter.

diverticulum makes it impossible for this head of pressure to be maintained. The conditions become still more unfavourable when there is obstruction at the outlet. A case has been recorded of a patient who had a diverticulum with a considerable stone in it. He was able to micturate until the latter had been removed from the pouch by an operation; he then found himself deprived of this facility. The suggestion is that the removal of the calculus uncorked the diverticulum, which now filled with urine whenever micturition was attempted. *Case* 34 is of a similar type, for the patient had no residual urine until the calculi had been removed from his diverticulum, which was not resected. For the remainder of his life, which continued for several years, he was catheterized once a week, and his residual urine was always about 8 oz., and no doubt there was a further quantity of concealed residual urine. It is this lack of contractile power which causes the retention of some urine in the pouch, even after catheterization. In *Case* 26 (*Fig.* 578) there is still a small amount in the bladder cavity, but most is in the pouch (the other

shadows are thought to be glands—this patient has not yet been operated upon). This has been well called by Swift Joly, " concealed residual urine ". The quantity left behind in this manner after catheterization is never as much as the total capacity of the pouch and may be quite small, but the volume is of importance in deciding treatment. *Case* 30 (*Fig.* 579) is a further example of this. All cystographic medium had been drawn off by a catheter, which had then been removed before the photograph was taken.

Clearly it is this stasis which renders these patients so liable to *infection.* These 53 cases all had bladder sepsis except 3, and in 8 instances it was very severe. When once this is established no amount of bladder washing can cure it, and though sometimes it may be lessened by this means, in many instances no improvement results; indeed the only way to cleanse a really dirty diverticulum is, when the bladder has been opened, to run fluid into it through a catheter and withdraw it by means of a sucker.

FIG. 580.—*Case* 21. Large vesical calculus and a small one in a diverticulum.

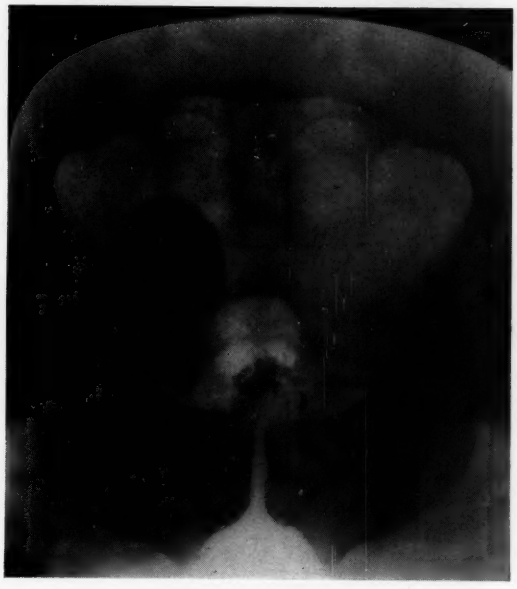


FIG. 581.—*Case* 22. A vesical calculus lying in an unusually lateral position.

Calculus Formation.—No doubt it is the stagnation already referred to which predisposes to the formation of calculi. In 13 cases this condition obtained; in 10 of these there was a calculus in the diverticulum as well as a stone in the bladder itself, in 2 there was a calculus only in the diverticulum, and in 1 case a stone was present in the bladder alone. Often it is easy from the position of a stone in a radiograph to be sure that it is outside the bladder itself, as in *Case* 21 (*Fig.* 580), in which the large stone was in the bladder and the smaller one in a diverticulum on the right side. But X-ray diagnosis is not always such a simple matter. The large stone in *Fig.* 581 lay in the bladder, whereas the stone in *Fig.* 582 (*Case* 22) was in a diverticulum. A doubt may occasionally remain even after cystoscopy, though this will sometimes show a portion of a calculus protruding from a

diverticulum, as happened in this example. If therefore litholapaxy is performed, a radiograph should be taken soon afterwards; or when lithotomy is performed, if all shadows have not been accounted for, the orifice of a diverticulum must be

FIG. 582.—*Case* 22. Calculus in a diverticulum. The beak projects towards the bladder cavity. Prostatic calculi.

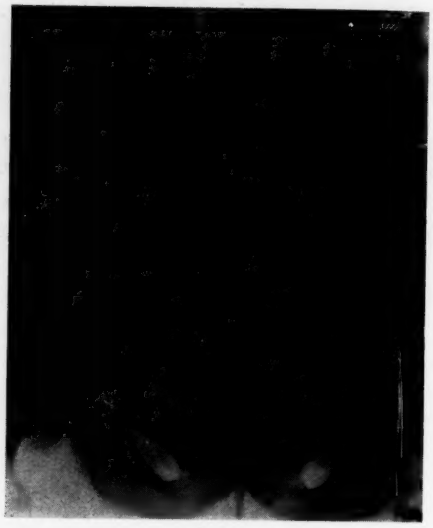


FIG. 583.—*Case* 22. Cystogram after the calculus has been removed. The sac was not excised.

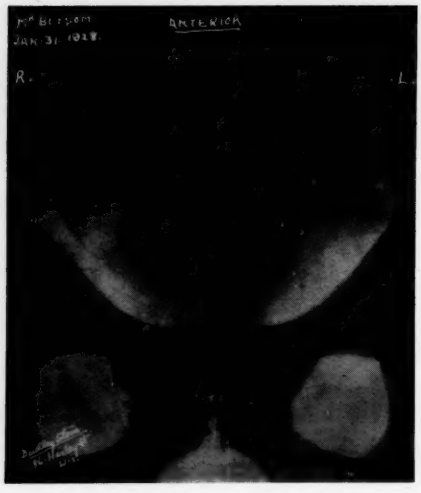


FIG. 584.—*Case* 34. Calculi A and C were in a diverticulum, B in the bladder cavity. Prostatic calculi.

FIG. 585.—*Case* 48. The long calculus was in the bladder. The large round one and the two smaller ones were in a diverticulum.

sought. The calculus in *Case* 22 (*Figs.* 582, 583) had been missed when a lithotomy had been done elsewhere one year previously. I removed it by slitting sufficiently the orifice of the sac, which itself was not excised. The next case, *Case* 34 (*Fig.* 584),

had a similar history. He was a very sick man of 73 who had suffered from cystitis for six years. One year before I saw him, his prostatic urethra had been dilated (he had prostatic calculi), and litholapaxy had been attempted, presumably

FIG. 586.—*Case* 47. Vesical calculi and a group of calculi in a diverticulum.

because of the X-ray appearances, but no stone could be gripped. Using a flushing cystoscope I was able to assure myself that only one stone was free in the bladder, and on cystotomy it was extracted and the others removed from a diverticulum

FIG. 587.—*Case* 47. Concealed residual urine obscuring calculi in a diverticulum.

after incision of its opening. In a younger subject diverticulectomy would have been carried out, for without this there was an obvious likelihood that further stones

would form in the pouch. However, he considered himself sufficiently cured; but as has already been stated earlier in this paper, for the remainder of his life he was catheterized once weekly and washed out, and there was always a residuum of about 8 oz. of septic urine.

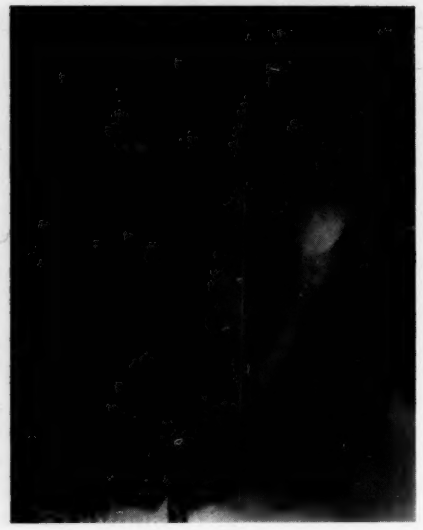


FIG. 588.—*Case* 19. Opaque ureteric catheter coiled up in a diverticulum.

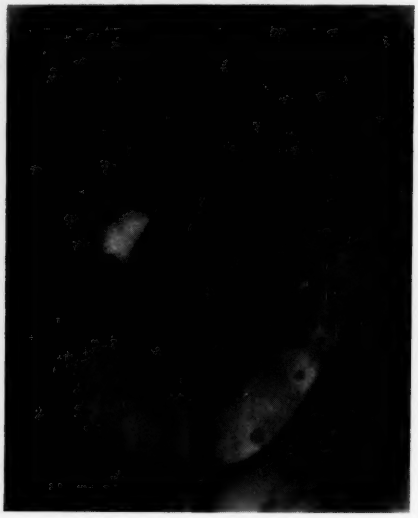


FIG. 589.—*Case* 19. Sodium iodide injected.

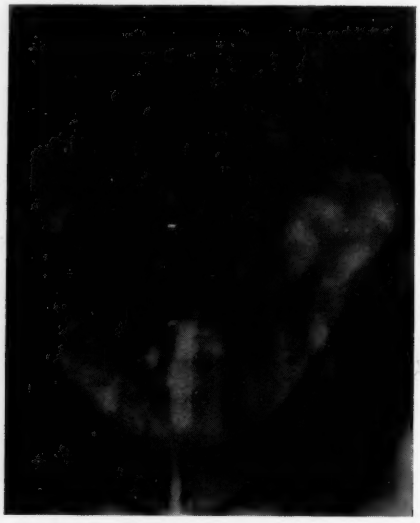


FIG. 590.—*Case* 19. Cystoscope withdrawn after emptying the bladder. Concealed residual urine.

In another, *Case* 48, prostatectomy in two stages had been performed for retention three years before I saw the patient, but he stated that he had never been comfortable, and latterly had had pain in the perineum which prevented him stooping down to fasten his shoes. He had also begun to pass stones within a few

months of recovering from the operation. There was severe cystitis. Lithotomy revealed only one calculus, the long one, but the openings of many sacculi were seen, and in a diverticulum which opened above and external to the right ureteric orifice was found the other large round stone (*Fig.* 585) lying quite loose in it, together with three smaller ones. The diverticulum was excised, but another not so large, on the opposite side of his bladder, had to be left untouched. He was well for nearly two years after this, except for two attacks of cystitis, but then his kidneys became acutely infected and he died of uræmia. It seems quite certain that these diverticula, and probably the stone in the largest of them, had been present long before the prostate had been removed, yet this operation had been done by an experienced urologist. This case also illustrates one of the disadvantages of performing prostatectomy in one or two stages without a careful examination of the bladder.

It may be thought that if an open operation is performed a diverticulum is unlikely to be missed, but this is by no means a certainty, for in *Case* 47 (*Fig.* 586). I knew from a previous cystoscopic examination that three medium-sized and two small stones were present in the bladder and I had seen the orifice of a diverticulum very close above that of the right ureter containing other small stones. On opening the bladder I easily found the stones lying in it, but could not locate the opening of the diverticulum without wide retraction and a careful search with the points of a pair of curved scissors, and this in spite of the fact that I knew exactly where to look for it. *Fig.* 587, also a radiograph of this patient, shows the appearances after as much as possible of the sodium iodide solution had been voided, and indicates the volume of concealed residual urine, the shadow of which still obscures the calculi. It would not be wise to say that cystoscopy should be performed on every patient requiring operative treatment for prostatic obstruction, but it is certain that this method of examination is by far the most accurate means of determining the existence of a diverticulum of the bladder, particularly if a flushing cystoscope is used, as by this means the bladder can gradually be distended, thus bringing into view the dark opening of the diverticulum, which may have remained closed when the bladder was less fully distended.

The passage of a ureteric catheter into the diverticulum may be of assistance in determining its size. *Case* 19 (*Fig.* 588) illustrates this. In *Fig.* 589 the cystographic material has been run into the bladder through the cystoscope, but *Fig.* 590 again exemplifies the more accurate means of determining the size of any diverticulum, namely cystography. In this, a radiograph of the same patient, the bladder has been emptied by catheter, and the concealed residual urine is seen to remain in the diverticulum.

A Neoplasm in Relation to a Diverticulum.—There are four cases of a neoplasm in relation to a diverticulum to report. The first, *Case* 3, was given in the previous paper. There had been hæmaturia five years previously, and again one year before operation. It was found that the spread of the growth outside the bladder had involved the wall of the diverticulum by extension from without. The operation, partial cystectomy which included the pouch, was performed in 1923, and he still remains alive and well. The next, *Case* 37, was aged 62. He had passed blood-stained urine three times in the previous twelve months, and had noticed his stream to be somewhat weak, and there were 12 oz. of residual urine which was infected. *Fig.* 591 shows the bladder and diverticulum distended with

urine rendered opaque by uroselectan, which had been injected intravenously four hours previously. Slight dilatation of the left ureter is seen. The diverticulum appears to be chiefly in the left side of the pelvic cavity, but actually its orifice was some little distance posterior to that of the right ureter. *Fig.* 592 gives the appearance

FIG. 591.—*Case* 37. Cystogram obtained by intravenous uroselectan. The diverticulum is a dense shadow of smooth outline lying behind that of the bladder, which is irregular. Left ureter slightly dilated.

FIG. 592.—*Case* 37. After micturition.

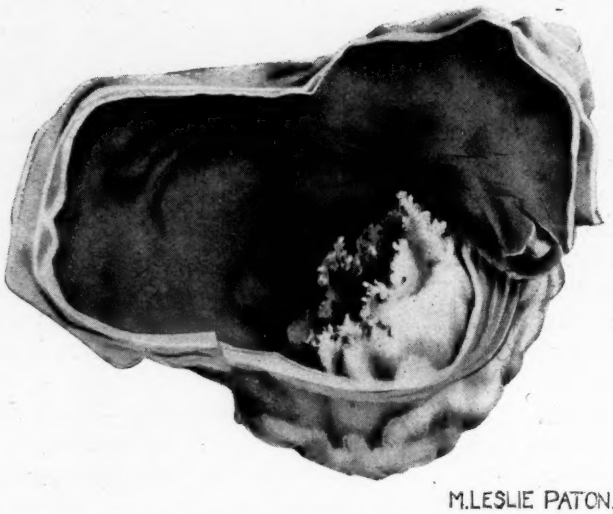


FIG. 593.—*Case* 37. A diverticulum seen from within, a papilloma being immediately below the vesical orifice.

after micturition, and illustrates how very imperfectly these patients empty their bladders. On cystoscopy the opening of the diverticulum was not apparent with 8 oz. of fluid in the bladder, and became only slightly opened when this volume had been increased to 16 oz., and had it not been that the bladder was free from

sacculation and trabeculation, its presence might have remained undetected. On diverticulectomy the presence of a growth was recognized. It was a villous papilloma; its exact nature was uncertain, but it was perhaps malignant. *Fig.* 593

FIG. 594.—*Case* 25. Cystogram. The bladder had been emptied. The mesial aspect of the diverticulum is relatively translucent owing to the presence of an epithelioma.

FIG. 595.—*Case* 25. The halves of a diverticulum laid open through its orifice and containing a carcinoma and a calculus.

shows the diverticulum after it had been resected, and the growth within it. Although there was a small intravesical enlargement of the prostate, this has not so far been treated.

The third example, *Case* 25, was a patient aged 58 who complained of painful micturition and occasional slight hæmaturia, and for one year the urine had contained blood and pus. The growth was an epithelioma with cell-nest formation; calculi were also present in the diverticulum, which opened near the left ureteric orifice. *Fig.* 594 shows the sodium iodide solution left in the diverticulum after the bladder had been emptied. It will be seen that the shadow is most dense in its outer part and relatively translucent near the middle of the pelvis. This appearance was produced by the solid structure of the growth within the diverticulum, which caused a filling defect in this part of it. *Fig.* 595 depicts the diverticulum laid open in two halves after resection.

Case 37 was very well two years and five months after the operation, and *Case* 25 was seen to be in very satisfactory condition and free of symptoms two years and two months after the operation.

The fourth example, *Case* 52, was a patient, aged 58, who had suffered from hæmaturia on two occasions during the last two years. On cystoscopy a papilloma of a benign type, measuring about 3 cm. across, was found obscuring the left ureteric orifice. It was not pedunculated, and extended up to the orifice of a diverticulum situated above and behind the left ureteric orifice. This diverticulum was a small one, about 4 cm. deep. The treatment undertaken was diathermy of the papilloma, and when this had been cleared away it appeared that there was no extension of the growth to the interior of the sac. The case remains under observation.

FIG. 596.—*Case* 36. A large diverticulum on the right, a lesser one on the left. A small amount of opaque fluid remains in the bladder below.

FIG. 597.—*Case* 36. Excretion urogram showing right nephro-hydro-ureter, and renal calculi which formed in the left kidney after diverticulectomy.

Dilatation of the Ureter.—Ureteral dilatation coincident with the presence of a vesical diverticulum has been alluded to, and may be due only to general back-pressure in the urinary tract, but in most cases where it exists it is caused by the direct pressure of the diverticulum on the ureter where the latter approaches the

bladder. *Case* 36 illustrates this. *Fig.* 596 shows a very large diverticulum on the right side of the bladder, and a smaller one on the left. The patient was 57 years old and of poor physique. He was very ill, his urine being foul and stained with blood. He had suffered from difficulty in micturition for five months, and his bladder was now distended to the umbilicus. Radiography showed that there were stones in the diverticulum which was on the right side. Uroselectan before operation showed the right ureter to be much dilated throughout its extent, and gross hydronephrosis of the calices above. After a fortnight with a tied-in catheter, during which attempts were made to cleanse the bladder, it was explored. The large diverticulum was excised and also the small one, the latter by cutting round it and invaginating it, the former by first dividing the bladder down to its orifice. Intense sepsis followed with much sloughing, and nearly two months later a further operation had to be carried out. It was found that on the right the bladder had broken down completely and a large area of the pubic bone was exposed. The vesical wall had to be freed from this before it could be repaired. Fortunately the right ureteric orifice was not involved. One month later he was ready for a perurethral resection, there being a slight enlargement of the prostate, and this was successfully performed. Subsequently he considered himself well, and is at work again, but further excretion urograms (*Fig.* 597) show that in addition to the dilated right ureter, he had, in the space of eight months, grown multiple calculi in the left kidney where none had existed when he was first investigated.

DIAGNOSIS

This has already been fully discussed in describing individual cases, but in summarizing it may be pointed out that the recognition of a vesical diverticulum is most conclusively established by cystoscopy, and that next in value as a means of diagnosis is cystography. It is rare that the condition can be recognized on general examination, but there are two cases in this series in which the provisional diagnosis was made by this means. In one, *Case* 44, the patient had himself noticed a swelling in the right side of the lower abdomen, but the practitioner recognized that there was also one on the left, though this was less well defined. The former was the bladder, the latter a diverticulum. Fifty-two ounces of urine were withdrawn by catheter. In *Case* 46 the conditions were similar. I was able to recognize the distended bladder on the right and a distinct tumour on the left of the lower abdomen. In this connection it is useful to remember that although the tumour formed by an over-distended bladder is usually in the middle line, it quite frequently rises up beneath the right rectus muscle; only rarely does it appear in the left half of the abdomen.

TREATMENT

When sacculations of the bladder wall are observed on cystoscopy the cause must be carefully sought for. This may be an enlargement of the prostate or a urethral stricture. A less common, but very important, cause is the condition known as Marion's disease, or stenosis of the col vesicæ. It is, of course, very well recognized that these conditions require treatment, and this may be considered preventive of the formation of diverticula. When once, however, a sac of any considerable size exists, such treatment alone, though of the greatest importance, is insufficient for cure.

If sepsis is established, it is extremely difficult to combat it successfully, and although a patient's condition may be somewhat improved by bladder irrigations, it can never be eradicated by this means alone, for the reason that it is impossible adequately to wash out a diverticulum by any transurethral method; indeed this is difficult enough, as has already been stated, even when the bladder is laid open.

Perurethral Prostatic Resection.—Attempts have been made to treat cases of vesical diverticulum associated with prostatic obstruction by means of perurethral prostatic resection alone. In this series I employed this procedure in 4 instances, and all were unsuccessful, though in the first, *Case* 41, the method should probably not have failed. This patient, aged 47, had two small diverticula near the right ureteric orifice. Prostatic calculi were also present, and I judged that the obstruction was due to changes in this gland. Perurethral resection was performed, with the aim of releasing sufficient of the calculi for the relief of this obstruction. Eleven days after leaving hospital he developed fever and dyspnœa, and a septic swelling appeared in the neck. He was re-admitted, and died, twenty-one days after the resection, of cellulitis and right lobar pneumonia. Although it is impossible to be certain, I cannot help feeling that his death was the outcome of the operation.

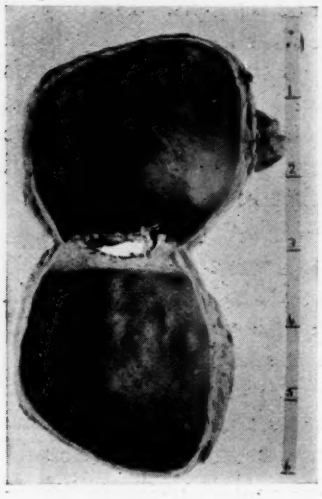


FIG. 598.—*Case* 30. An excised vesical diverticulum laid open.

Perurethral resection was next used in *Case* 30, a patient, aged 80, who had suffered from painful micturition for ten years. He had retention overflow, the top of the bladder being level with the umbilicus and containing two pints of urine. He had a diverticulum much larger than the bladder itself opening above and external to the right ureteric orifice (*see Fig.* 575). Treatment by daily catheterization did not succeed in re-establishing micturition; moreover much concealed residual urine was also present. The prostate on rectal examination was small and of firm consistency; bimanually, however, a considerable intravesical enlargement could be palpated, and this was confirmed on cystoscopy. In the hope of sparing the old man a severe abdominal operation, I decided to employ the method of perurethral resection; this, however, failed entirely to relieve his retention. *Fig.* 576 is a lateral view taken nineteen days after this, and shows how useless the operation had been. Indeed, had such a demonstration of the character of the pouch been made when I was first investigating the case, I should have realized the futility of attempting anything less than diverticulectomy. The operation was not merely followed by failure, but also by an intense degree of bladder sepsis, and the urine, which had previously been clean, became very foul. A temporary pyuria is well known often to result from the treatment of prostatic obstruction by diathermy, but in the present case the intensity of it was extraordinary, being encouraged by the stasis of the contents of the diverticulum, which quickly became infected and helped to maintain the cystitis. I therefore excised the diverticulum (*Fig.* 598), which is seen in antero-posterior view in *Fig.* 579.

At the operation I had a good chance to study the amount of resection of the prostate which had been effected, and this seemed to me at the time to have been

quite adequate—all of the intravesical projection had been removed in the region of the trigone and up to 2 o'clock on one side (as seen in a cystoscopic view) and up to 9 o'clock on the other. However, although the suprapubic fistula closed, it broke down on several occasions after the catheter had been removed, and three weeks later I removed the small remainder of his prostate by enucleation. Following this his recovery was uninterrupted, and seen eight months later he was extremely well, and his urine was perfectly clear.

In the third instance, *Case* 24, in which I have employed perurethral resection, the operation was more satisfactory in its first effects. The patient was 65 years old, and complained of difficult and frequent micturition, which he said he had noticed for only three weeks. His bladder was distended to the umbilicus and he had more than one pint of residual urine. Per rectum his prostate was found

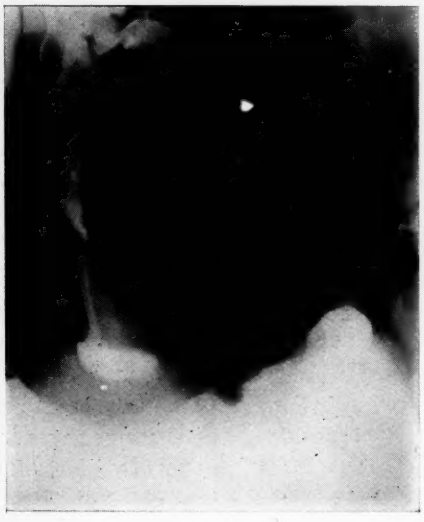


FIG. 599.—*Case* 24. Oblique cystogram. The diverticulum lies above and posteriorly. Before operation.

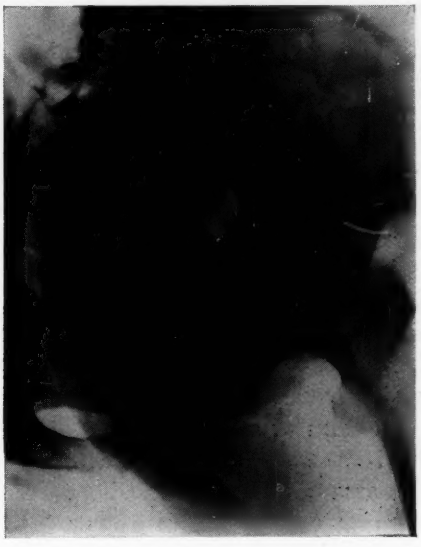


FIG. 600.—*Case* 24. Cystogram showing concealed residual urine after perurethral resection.

to be small, but elastic. On cystoscopy a definite intravesical enlargement could be seen, and also the opening of the diverticulum behind the interureteric bar, as shown in *Fig.* 599, which is an oblique cystogram. Perurethral resection was carried out, and *Fig.* 600 shows the appearances nine days later. After filling the bladder with sodium iodide the catheter was withdrawn and the patient instructed to micturate; it will be seen that the diverticulum now empties almost completely. The residual urine when tested three weeks after the operation had dropped to 2 oz. A fortnight later, however, the amount had risen to 4 oz., and a week later to 6 oz. For a year he considered himself in a satisfactory condition, but then an attack of acute retention of urine developed, requiring catheterization, and subsequently micturition was difficult and the urine very septic. Therefore diverticulectomy was performed, this being thirteen months after the first operation; at the same time the prostate was examined and it was decided not to remove the residuum of it. Following this he did well, and his condition eight months later was satisfactory, though there were still 4 oz. of somewhat septic urine, and it may be that further treatment of the prostate will become necessary.

In *Case* 35 severe cystitis resulted, and diverticulectomy was necessary two months later for cure.

In criticism of these cases it might be argued that the resections were inadequate —and in one case this clearly was so—but the chief objection to adopting this method when vesical diverticula are present is that it inevitably establishes a very serious degree of sepsis, both in the bladder and still more so in the diverticulum, which cannot be eradicated by anything less than diverticulectomy. When, however, diverticulectomy has been carried out, perurethral resection of the prostate may be a valuable method of treating obstruction at the vesical neck. With this object it was used in 6 cases in this series besides the 4 instances just described.

Excision of the Diverticulum.—The more radical forms of operation are undertaken by the suprapubic route, and should aim at the excision of the diverticulum.

Laying Open the Diverticulum into the Bladder.—Attempts are sometimes made to produce a cure by merely laying open the diverticulum into the bladder itself. In this series there are two examples in which this had been done.

One of them, *Case* 23, a man aged 54, had suffered from increasing difficulty in micturition for two years, and had been operated upon elsewhere six months previously, a diverticulum having been recognized. The surgeon had been content merely to enlarge its orifice and then to suture the edges afresh to the bladder. Since then the patient had never passed any urine naturally, and it had become intensely septic. The cystoscopic appearances were astonishing; the bladder itself was so contracted that it was a difficult matter to find it at all, whereas the vast expanse of the diverticulum appeared as a glistening white vault, some parts of which were covered with muco-pus. It is easy to understand how micturition was impossible, for as a result of the operation his bladder became like the half-closed palm of a hand, and its grip upon the urine in contact with it was as little effective. The left ureteric orifice was ultimately identified, but the right was lost in an area of intense congestion and œdema at the junction of the two cavities. The operation, which occupied four hours, consisted in the freeing and excision of the diverticulum and the re-formation of the bladder, and happily it was found possible to restore the right ureteric opening into a normal position. Although no enlargement of the prostate could be certainly recognized, perurethral resection of the gland was carried out three weeks later, and when the cystotomy had closed, the residual urine was reduced to less than 1 oz., and has so remained.

In the other example, *Case* 43, a similar operation had been performed in another country twenty-two years previously, and at the time of examination he had reached the age of 69. Since the operation the amount of residual urine had never been less than 8 oz., and this had been persistently septic in spite of a long period of bladder washing. However, he had continued to live an active life and was in the main well.

It may be thought that I have laid undue emphasis on the importance of dealing with *prostatic obstruction* which is so little obvious by either digital or cystoscopic examination. In one of my first cases of diverticulectomy, *Case* 2, the operation was successfully completed, but the patient's suprapubic fistula remained open for seven weeks, although it had been closed more than once by catheter drainage. By rectal examination I had considered his prostate to be normal, and cystoscopy had confirmed this view, though possibly nowadays with the aid of a

cysto-urethroscope I might think differently. Something more had clearly to be done, and I re-opened his bladder. A colleague who was present agreed with me that there was no intravesical enlargement of his prostate. However, I put a finger into the internal meatus, but failed to find any plane of cleavage. Then I took off my glove and thus unhampered quickly enucleated two very small lateral lobes, which were later reported as " chronic prostatitis with glandular hypertrophy ". After this he quickly healed, and remained in good health until he died twelve years later of another disease. The relationship between diverticula and degrees of prostatic obstruction which are not easily recognized has interested me ever since this case.

Diverticulectomy.—In performing the operation of diverticulectomy it is necessary to know the number, position, and size of the sacs. This information must be obtained by a preliminary cystoscopy and by radiography. When once the bladder has been opened, it is sometimes not easy to find the diverticulum, even with the help of special self-retaining retractors. If the orifice of a diverticulum which is to be removed is at all near that of a *ureter*, it is sound practice to pass a rather stiff bougie up the latter to the kidney, and to leave it there throughout the operation. By this means injury to the duct is usually avoided, or if in spite of this precaution the latter does get damaged, as occurred in *Case* 47, where its wall was partially divided just outside the bladder, repair is more easily carried out, the bougie being a guide to its situation.

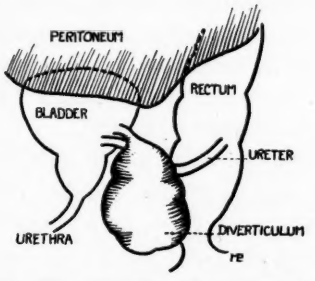


FIG. 601.—*Case* 39. Diagram illustrating the usual relationship of the ureter to a lateral diverticulum.

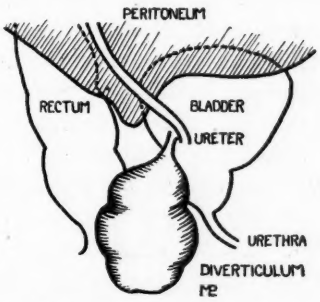


FIG. 602.—*Case* 24. Diagram illustrating an uncommon relationship between the ureter and a diverticulum.

The ureter is always closely related to the neck of any medium-sized or large diverticulum which takes origin from near the upper angles of the trigone. The duct lies usually between the sac as this dips down towards the bottom of the pelvis, and the bladder—that is, below the neck, as is shown in *Fig.* 601 (*Case* 39). Much less often the ureter passes above the neck, as in *Case* 24, this condition being depicted in *Fig.* 602.

The resection of a diverticulum is a very simple matter when it is not large and lies away from the ureters in the fundus of the bladder, as in *Cases* 26 and 45, or near the apex, as in *Case* 27. These are the easiest cases; even here care is necessary, and attempts to drag the bottom of the sac into the bladder by means of catch forceps or by pneumatic suction are to be condemned, as there is a risk of pulling upon the peritoneum, or even upon its contents. Diverticula can be dealt with by a transvesical approach provided they are not large—that is to say, after the bladder has been opened, a circular cut is made round the orifice with a knife,

including all the coats of the bladder. Catch forceps are then applied to the detached edges, and while pulling upon these, and sometimes using a finger within the sac, a dissection is carried out with curved scissors to free it on all aspects and thus gradually it is drawn into the bladder cavity. This dissection is always most difficult near the bottom of the pouch, for here it is usually particularly adherent. If the case is strictly aseptic the hole in the bladder wall may be sutured; if not, a strip of corrugated rubber should be pushed through it into the space behind the bladder, the other end of it being allowed to protrude from the abdominal wound, which is closed in the usual manner around a cystotomy tube. This rubber can be removed after a few days, and the incision in the bladder wall from which it has been withdrawn will heal soundly.

When, however, the diverticulum is a large one, I prefer to approach it by the following method: The bladder is opened by a small incision, which is subsequently carried down its lateral wall to the neck of the diverticulum and then around its orifice. When this has been done the bladder falls away towards the opposite side of the pelvis and approach to the sac is easier. It is dissected free with long curved scissors, and as an aid to this a finger within its cavity is very helpful. It should be realized that it is only necessary to remove its innermost layer, and that often several planes of cleavage are found during the exposure. In most cases I have removed the diverticulum intact. *Fig.* 598 shows such a specimen with the orifice in the centre. But no harm is done if it is torn during the process; indeed it is not essential even to remove the whole of it. Sometimes the bulk of it is torn away from the deepest portion, for this may be very far down in the pelvis, being often in the neighbourhood of the ischial spine and very adherent. Not infrequently there is troublesome bleeding at this stage which needs to be controlled, and always some oozing remains. Having satisfied oneself that the ureter is intact, the lateral incision in the bladder is repaired by a series of interrupted sutures through all its coats, the knots being tied on the external surface. Care is taken not to compress the ureter by any suture too close to its orifice; at this stage the ureteric bougie is removed. The bladder is drained by a suitable cystotomy tube, and another small tube is laid outside the viscus down into the depths of the bony pelvis, the two being brought out together through the abdominal incision. In most cases there is very little subsequent bleeding, and although abdominal distension is sometimes troublesome, convalescence is usually rapid.

If the *ureter opens into the diverticulum*, as it did in *Cases* 7, 44, and 46, it will be necessary to re-implant it, and this is done by attaching it to the incision in the bladder wall. Two sutures of fine plain catgut are usually sufficient, and the procedure does not differ from that used in similar operations for malignant growths of the bladder. It was successfully adopted in *Cases* 7 and 44; in *Case* 46 cystotomy only was performed, and the patient died from pyonephrosis before any radical operation could be undertaken. In two other instances, *Cases* 12 and 37, the opening of the ureter was so close to the orifice of the diverticulum that it was detached during the resection, and for this reason had to be re-implanted. In the former, *Case* 12, the patient died of pyelonephritis; in the latter, *Case* 37, the result was entirely satisfactory.

When the condition is associated with an enlarged prostate, treatment of this must be undertaken; and if enucleation is the method chosen, this can be performed at the conclusion of the diverticulectomy, if the patient's condition should be

satisfactory. Otherwise it is better postponed for about a fortnight, and then undertaken as a second-stage operation. This order should never be reversed, for if prostatectomy is first performed, the resultant scarring in the region of the abdominal wound, and the collapse of the bladder consequent upon its drainage, will altogether render diverticulectomy a much more formidable procedure.

In this series of 53 cases diverticulectomy alone was performed in 15 instances, diverticulectomy combined with treatment of prostatic obstruction either by prostatectomy or perurethral resection in 21 instances, making a total of 36 diverticulectomies. There was one immediate death following diverticulectomy (*Case* 12). One patient, *Case* 4, died after a second-stage prostatectomy. Two patients, *Cases* 13 and 46, died after cystotomy. One, *Case* 40, in which multiple small diverticula existed but were not treated, died because one of these was ruptured when using a Bigelow's evacuator after a vesical calculus had been crushed. Litholapaxy is never employed when there are diverticula of large or moderate size, because of the obvious impossibility of recovering the fragments of the calculus; this case is included in order to show that in the presence of even small pouches litholapaxy carries a risk which is not so well recognized. *Case* 41 died three weeks after perurethral resection of the prostate. *Case* 29 died three months after diverticulectomy and prostatectomy. *Case* 48 died twenty months after diverticulectomy. *Case* 22 died one year after diverticulectomy of cardiac failure. *Case* 18 died four and a half years after diverticulectomy and prostatectomy of apoplexy. *Case* 2 died twelve years after his operation of another disease. The other patients, as far as can be ascertained, are all alive and well.

CASE SUMMARY

All patients are males with the exception of *Case* 51. The first eleven case descriptions in this series of 53 cases refer to those which were discussed in "A Clinical Study of Eleven Cases of Vesical Diverticula" published in the BRITISH JOURNAL OF SURGERY (1925, xiii, 144), but which contained no summaries.

An attempt has been made to determine *the condition in each case which led to the formation of the diverticulum.* This is stated at the end of each case note under the heading 'Causation'.

Case 1.—J. M., age 65. Complaint: Bladder pain for eighteen months and difficulty of micturition, retention of urine four months previously; also aching pain in the left hip and back of thigh. Residual urine 15 oz. Large diverticulum with opening near left ureteral orifice. No prostatic or other obstruction. Operation: Diverticulectomy. Twelve years later very well, except left sciatica troublesome since four years after the operation. *Causation*: Undetermined.

Case 2.—W. M., age 60. Complaint: Increased frequency of micturition for five years, with difficulty in starting and poor stream; short history of pain in left groin and perineum. Residual urine 25 oz., containing pus and *B. coli.* Large diverticulum opening behind the interureteric bar close to midline; a very small one beside it. Operations: Both diverticula excised; subsequent excision of fibrous prostate necessary. Died without further urinary symptoms twelve years later. *Causation*: Prostatic obstruction.

Case 3.—F. B., age 61. Complaint: Hæmaturia five years previously, recurrence during last twelve months, difficulty and frequency of micturition. Residual urine 5 oz., infected. Diverticulum above and outside right ureteral orifice, with a carcinoma at its opening. Operation: Partial cystectomy and diverticulectomy. Twelve years later residual urine 1½ oz.; no recurrence of growth or diverticulum. *Causation*: Undetermined.

Case 4.—W. T., age 63. Complaint: Hæmaturia, hourly frequency with painful and difficult micturition. Stream poor. Internal urethrotomy nine years previously. Residual urine 1 oz., very heavily infected. Operations: Calculi removed from diverticula situated above and external to each ureteral orifice; three weeks later prostatectomy for fibrous prostate. Died. *Causation*: Prostatic obstruction.

Case 5.—J. G., age 58. Complaint: Difficulty of micturition and for two years frequency and recent severe nocturnal incontinence; a perineal fistula for six months. One large and one small diverticulum above and external to right ureteral orifice and a fibrous prostate. Operations: Excision of fistula; one month later excision of diverticula. Three months later secondary prostatectomy. Nine years later free of symptoms. *Causation*: Prostatic obstruction.

Case 6.—J. P., age 73. Complaint: Acute retention, preceded by severe frequency of micturition and pain for five months. Urine infected. Medium-sized diverticulum behind interureteric bar on right; considerable enlargement of prostate. Operation: Prostatectomy. Apparently cured, but no subsequent history available. *Causation*: Prostatic obstruction.

Case 7.—V. E., age 15. Complaint: Painful micturition for two years, hæmaturia three times in six months; no frequency, stream good; urine contained blood and was infected. Large diverticulum in the position of left ureteral orifice. Operations: Diverticulectomy and transplantation of ureter. Cured, but no subsequent history available. *Causation*: Congenital abnormality.

Case 8.—L. S., age 45. Complaint: Considerable frequency of micturition for many years, with a poor stream; a meatal stricture for thirty years. Residual urine 2 oz., with much infection. A vesical calculus present, and a diverticulum above and internal to left ureteral orifice, with a mushroom calculus in it. Operations: Litholapaxy. Six months later a calculus removed from the bladder and diverticulectomy. Well seven years later, except frequency and *B. coli* infection. Cystoscopy: Very shallow diverticulum above and external to right ureteral orifice; cystitis; the region of left ureteral orifice normal. *Causation*: Meatal stricture.

Case 9.—E. J., age 63. Complaint: Difficulty of micturition. Catheter life for five weeks, residual urine 13 oz., infected; enlarged prostate. A large diverticulum above and internal to left ureteral orifice. Operation: Diverticulectomy. Cured, but no subsequent history available. *Causation*: Prostatic obstruction.

Case 10.—J. B., age 45. Complaint: Partial retention of urine for six months with frequency of micturition, urine infected, urethral stricture, two diverticula present, spina bifida occulta of 5th lumbar vertebra. Operation: Cystotomy for sepsis, and eight months later excision of diverticula. Improved. *Causation*: Urethral stricture.

Case 11.—W. B., age 69. Prostatectomy had been performed two years previously for prostatic calculi. Complaint: Frequency of micturition and severe difficulty, no residual urine, infected; several small diverticula on bladder base. Operation: Suprapubic lithotomy; the diverticula were not excised. *Causation*: Prostatic obstruction.

The following cases were not discussed in the previous paper on this subject:—

Case 12.—D., age 43. Operation had been performed on right kidney by another surgeon, resulting in a renal fistula. Bilateral renal calculi were present, also a stone in a large diverticulum close to the right ureteral orifice and obstructing the duct. There was a small diverticulum above the left ureteral orifice. Acute cystitis was present. Operation: In excising the diverticulum and freeing the right ureter it was necessary to transplant the latter. The patient died of pyelonephritis. *Causation*: Undetermined.

Case 13.—H. R. S., age 69. Complaint: Frequency of micturition for two years, incontinence of urine for one year. The bladder was distended, the urine infected, and the prostate enlarged. Small diverticulum with left ureteral orifice opening on its lower margin. Cystogram: Reflux up much-distended ureter to left hydronephrosis. Operation: Cystotomy. The patient died. *Causation*: Prostatic obstruction.

Case 14.—E. H. H., age 48. Complaint: Difficult, frequent, and painful micturition for six years. Residual urine 2½ oz. Two moderate-sized diverticula, one the size of a hen's egg above and external to right ureteral orifice, a larger diverticulum above left ureteral orifice. Operations: Cystotomy for severe cystitis; subsequent diverticulectomies and diathermic coagulation of small intravesical enlargement of prostate. Residual urine two and a half years later 1 oz.; four years, 1 oz. Two years later residual urine 5 oz., not infected. On cystoscopy: Two very shallow diverticula now present in sites of those excised, no obvious enlargement of prostate. Operation: Perurethral resection. Six months later residual urine 1 oz. *Causation*: Fibrous prostate.

Case 15.—J. L., age 72. Complaint: Hæmaturia, difficult micturition for three years. Residual urine 2 oz., infected. Two small diverticula above and external to each ureteral orifice confirmed by cystograms. No operation. *Causation*: Prostatic obstruction.

Case 16.—W. L., age 62. Complaint: retention overflow with slight hæmaturia. Residual urine 50 oz., infected. Large diverticulum above right ureteral orifice. Operations: Excision; a fortnight later prostatectomy for moderate enlargement of gland. Eight years later very well, urine clean. On cystoscopy: No recurrence of diverticulum, no residual urine. *Causation*: Prostatic obstruction.

Case 17.—G. E. G., age 68. Complaint: Difficulty of micturition. Catheter life for six weeks. Urine infected. Three diverticula—a large one outside right ureteral orifice, one 2 in. deep on posterior wall, a small one on left lateral wall; small intravesical enlargement of prostate. Operation: Excision of all three diverticula, combined with prostatectomy. Seven years later very well, urine clear. *Causation*: Prostatic obstruction.

Case 18.—W. P., age 71. Complaint: Frequency and difficulty of micturition, and hæmaturia on two occasions six months ago. Residual urine 16 oz., infected. A diverticulum near right ureteral orifice and several smaller diverticula elsewhere. There was one stone in the largest diverticulum and two others in the bladder. Operations: Excision of largest diverticulum and prostatectomy three weeks later. Well until he died, four and a half years later, of apoplexy. *Causation*: Prostatic obstruction.

Case 19.—R. J., age 60. Complaint: Frequent, painful, and difficult micturition. Residual urine 3 oz., infected with *B. coli*. Lithotomy had been performed fourteen years previously. One medium-sized diverticulum above and external to right ureteral orifice. Operations: Excision. Infection and symptoms persisted, therefore four years later the fibrous prostate was removed. One and a half years later well, urine clear, residual urine ½ oz. *Causation*: Fibrous prostate.

Case 20.—J. W., age 43. Complaint: Painful and difficult micturition with poor stream. Considerable residual urine necessitating a period of catheter drainage. Urine infected and blood-stained. Operation: Lithotomy, a calculus the size of a greengage was removed from the bladder, another the size of a cherry was removed from a diverticulum near right ureteral orifice, this latter being excised. Three and a half years later residual urine 1 oz., and almost clear. On cystoscopy: A shallow recess near right ureteral orifice, and a similar one near left ureteral orifice. *Causation*: Probably a urethral stricture, for which he had been treated fifteen years previously.

Case 21.—E. F., age 32. Complaint: Frequency of micturition, for which he wore a urinal, also difficulty, and hæmaturia once recently. No residual urine, infected. A calculus protruding from a diverticulum near right ureteral orifice and one large stone in bladder. Operation: Diverticulectomy and removal of calculi. Six months later well. *Causation*: Undetermined.

Case 22.—T. R., age 60. Complaint: Prolapse of bladder wall through cystostomy incision following an operation one year previously for stone in the bladder. Operation: A calculus the size of a pigeon's egg removed from diverticulum above and behind right ureteral orifice, with diverticulectomy. Repair of abdominal wall, but cystostomy maintained. Well until one year later, when he died of cardiac failure. *Causation*: Obstruction due to prostatic calculi.

Case 23.—A. E. G., age 54. Complaint: Frequency and pain for two and a half years. Recent attacks of retention requiring catheter drainage. Residual urine 20 oz., infected. A previous operation performed elsewhere, apparently attempting to lay open the diverticulum into the bladder. Operations: Diverticulectomy, bladder being much contracted and the diverticulum huge. No prostatic enlargement obvious, but two months later perurethral resection of prostate. One year later well; residual urine 2½ oz., infected with *B. coli*. Two and a half years later well; urine slightly infected, residual urine 2½ oz. *Causation*: Prostatic obstruction suspected.

Case 24.—T. M., age 65. Complaint: Difficulty of micturition for three weeks and recent retention of urine. Residual urine more than 20 oz., sterile. A large diverticulum behind interureteric bar; slight intravesical prostatic enlargement. Operations: Perurethral resection, following which residual urine fell 2 oz., and micturition became free, painless, and easy, though the urine remained septic. One year later acute retention of urine, and cystograms showed the diverticulum as large as before, with much residual and concealed residual urine. Diverticulectomy therefore performed. Seven months later condition satisfactory; residual urine 4 oz. *Causation*: Prostatic obstruction.

Case 25.—E. M., age 58. Complaint: Pain in perineum one year, difficulty of micturition and slight hæmaturia. Urine infected *B. proteus*. A large diverticulum near left ureteral orifice. Operations: Diverticulectomy; the pouch contained several calculi, also an epithelioma. The fistula did not close, therefore subsequent perurethral resection performed, followed by slow but complete healing of wound. *Causation*: Undetermined.

Case 26.—W. B., age 63. Complaint: Suprapubic pain and difficulty in micturition, hæmaturia occasionally. Residual urine 12 oz., somewhat purulent, but sterile. A large diverticulum, the orifice being far back behind the right ureteral orifice, which probably contains calculi. Moderate enlargement of prostate. Has continued for three years without operation. *Causation*: Prostatic obstruction.

Case 27.—W. S., age 66. Complaint: Difficult micturition for six years. Residual urine 3 oz., not infected. Six years previously uric acid calculus passed from right kidney. Operation: A small diverticulum near the apex of the bladder resected, combined with prostatectomy. *Causation*: Prostatic obstruction.

Case 28.—B. D., age 51. Complaint: Frequent and painful micturition, hæmaturia once ten years previously. Residual urine 5½ oz., infected with *B. coli*. Two small diverticula far back behind each ureteral orifice. Operation: Both diverticula excised, combined with prostatectomy. Seven years later very well. *Causation*: Prostatic obstruction.

Case 29.—A. W., age 76. In very feeble condition. Complaint: Frequency of micturition for some years, and recent retention overflow. Urine infected with *Sta. albus*. Many small sacculations and one large diverticulum in the midline behind the interureteric bar. Operation: Diverticulectomy combined with prostatectomy. He survived the operation, but the residual urine never fell below 8 oz., and remained very infected. Died three months later of pneumonia. *Causation*: Prostatic obstruction.

Case 30.—W. H., age 80. Complaint: Painful micturition with frequency, and retention overflow. Urine very slightly infected. One large diverticulum close above and external to right ureteral orifice. Operations: Perurethral resection; the urine became very heavily infected and micturition was not re-established. Three weeks later, diverticulectomy. The perurethral resection was found to be complete posteriorly, but the intravesical enlargement of the prostate remained anteriorly. Ten days later perurethral resection repeated, but the fistula did not close. Six weeks later remainder of prostate enucleated; uninterrupted recovery. One and a half years later extremely well and urine quite clear. *Causation*: Prostatic obstruction.

Case 31.—C. V. S., age 50. Complaint: Frequent and painful micturition, and pain in right groin. Residual urine 10 oz., infected with streptococci and *B. coli*. Two moderate diverticula, one containing two small calculi above and behind right ureteral orifice. Operations: Both diverticula excised, combined with enucleation of prostate, the middle lobe only being hypertrophied. Three and a half years later very well. *Causation*: Prostatic obstruction.

Case 32.—J. N., age 58. Complaint: Difficult, very frequent, and painful micturition for two years. Residual urine 9 oz., much infected. Diverticulum very close to right ureteral orifice. Operation: Excision, combined with prostatectomy. Cured. *Causation*: Prostatic obstruction.

Case 33.—W. T. D. F., age 18. Complaint: Hæmaturia, and subsequently frequent and difficult micturition. Residual urine 8 oz., infected with *Sta. aureus* and *B. coli*. One large diverticulum near right ureteral orifice. Operations: Excision. Infection persisted, residual urine about 2 oz. Three and a half years later right nephrectomy; the kidney was very small and hydronephrotic, and the ureter dilated. Bladder infection persisted, *B. proteus*; and the residual urine varied from 1 to 2 oz. Three years later, because a cystogram showed a bulge on the right side of the bladder in the region of the previous resection, the bladder was explored, this area excised, and a partial resection of the bladder neck performed. Three months later much improved. *Causation*: Congenital abnormality.

Case 34.—W. B., age 73. Complaint: Painful and frequent micturition, with intermittence of stream. Residual urine nil. Severe cystitis. Very numerous prostatic calculi, medium-sized phosphatic calculus in bladder. Two larger articulating stones protruding into bladder from diverticulum behind and external to right ureteral orifice, and the orifice required wide incision to deliver the calculi. Survived several years, but residual urine about 8 oz. and much infected. *Causation*: Prostatic obstruction due to chronic prostatitis and prostatic calculi.

Case 35.—C. E. L., age 50. Complaint: Frequent, and difficult micturition for eighteen months, and pain in right renal area. Residual urine 9 oz., not infected. One large diverticulum above right ureteral orifice. A slight intravesical projection of prostate. Operations: Perurethral resection, severe cystitis resulted. Two months later, diverticulectomy. One month later, no residual urine; well. *Causation*: Uncertain, probably early prostatic obstruction.

Case 36.—C. C., age 57. Complaint: Difficult micturition for five months, and recent retention. Urine very septic. One large diverticulum below and external to right ureteral orifice containing calculi. One small diverticulum near left ureteral orifice. Operations: Diverticulectomies. Much sepsis resulted, requiring repair of bladder wall. Perurethral resection. Subsequently there was rapid increase of calculi in the left kidney. *Causation*: Prostatic obstruction.

Case 37.—G. C., age 62. Complaint: Hæmaturia two months; slight, frequent, and difficult micturition for two years. Residual urine 12 oz., infected. One large diverticulum above and behind right ureteral orifice, and one small one in the fundus. Intravesical enlargement of the prostate. Operation: Excision of both diverticula, the larger being found to contain a malignant villous papilloma; the right ureter was transplanted. Subsequent perurethral resection. One and three quarter years later condition satisfactory. *Causation*: Prostatic obstruction.

Case 38.—F. H., age 51. Complaint: Recent hæmaturia. A urethral stricture had been treated by bougies for seven years. Three weeks previously a cystotomy had been performed and papillomata excised; the presence of these appears to have prevented a successful search for a calculus seen in the bladder region by X rays. On examination: Cystostomy present, urine very septic, *B. coli* and diphtheroids. Operation: Fistula and scar excised, a sessile papilloma diathermied. Excision of diverticulum above and internal to left ureteral orifice exuding thick pus and containing the calculus. Cystoscopy two months later showed recurrence of papilloma, treated by cystoscopic implantation of radon seeds. Cured. *Causation*: Urethral stricture.

Case 39.—S. F., age 46. Complaint: Difficulty of micturition with poor stream for nineteen years, worse for two years. Micturition improved by exercising pressure on the abdomen above the right groin. Occasional hæmaturia. After micturition a cystic swelling was still palpable in hypogastrium. Residual urine 33 oz., infected with staphylococcus. A large diverticulum above left ureteral orifice. Operation: Diverticulectomy. Six months later condition satisfactory, urine clean. *Causation*: Undetermined.

Case 40.—F. W. D., age 70. Complaint: Frequent and painful micturition. Urine infected with *B. coli*. A large vesical calculus. Several small diverticula. Paget's disease. Operations: Litholapaxy. A week later X rays showed some small fragments remaining, therefore further treatment with Bigelow's evacuator. Two days' later cystotomy and peritoneal drainage for general peritonitis due to a rupture of a sacculus. Died. *Causation*: Prostatic obstruction.

Case 41.—H. R., age 47. Complaint: Frequent and painful micturition. Recent hæmaturia once. Urine infected with *B. coli*. Two small diverticula near right ureteral orifice. Prostatic calculi. Operation: Perurethral resection. Twenty-three days later returned to hospital with cellulitis of neck and bronchopneumonia. Died. *Causation*: Obstruction from chronic prostatitis and prostatic calculi.

Case 42.—J. H., age 54. Complaint: Acute retention of urine, relieved by catheter. Residual urine 2½ oz., infected with *B. proteus*. One large diverticulum above and external to left ureteral orifice. One small diverticulum above right ureteral orifice. Operations: Excision of both diverticula; three weeks later residual urine 3 oz., perurethral resection; nineteen months later condition satisfactory. Urine clean. *Causation*: Prostatic obstruction.

Case 43.—W. R. A., age 69. Twenty-two years previously operation for vesical diverticulum, apparently consisting in laying it open into the bladder. Since then persistent urinary sepsis, treated for many years by bladder washouts. Residual urine 9 oz., infected with *B. coli*. No enlargement of prostate. *Causation*: Undetermined.

Case 44.—R. B., age 64. Complaint: Slow micturition since boyhood: for two years stream poor and sometimes incontinence. For one year has noticed swelling in right side of lower abdomen. On examination two tumours here, that on the right side well defined, that on left side less so. Residual urine 52 oz., clean. Clinical diagnosis by his doctor: Vesical diverticulum. Large diverticulum at site of left ureteral orifice, which opened into it. Bougies catch at internal meatus. Operation: Diverticulectomy and partial excision of col vesicae. Six months later well. *Causation*: Stenosis of col vesicae.

Case 45.—G. W., age 48. Stricture of urethra treated by dilatation for ten years. Urine infected, recently very septic. Residual urine 1 oz. Two medium-sized and one small diverticula at bladder base, with other sacculi. Operation: Excision of three diverticula, which contained much thick pus. Eight months later very much improved. *Causation*: Urethral stricture.

Case 46.—C. W. M., age 67. Complaint: Nocturnal frequency and difficulty of micturition for ten months. Pain in lower abdomen, relieved by micturition, stream very poor. In a bad condition. A large tumour in left lower abdomen, and distended bladder palpable on right. Residual urine, a very large amount. Sterile. Three large diverticula, the left ureter opening into largest. Following catheter drainage very severe cystitis. Operations: Cystotomy. Much very thick pus from diverticula, tubes inserted. Died. Post-mortem examination: Left pyonephrosis, atrophic right kidney. A small intravesical enlargement of prostate of fibrous type. *Causation*: Prostatic obstruction.

Case 47.—H. Q., age 49. Complaint: Difficult and intermittent micturition, with pain. Residual urine 5 oz., infected. A medium-sized diverticulum close behind right ureteral orifice; five vesical calculi, and many more in diverticulum. Enlarged prostate. Operations: Lithotomy and diverticulectomy, and three months later second-stage prostatectomy. Cured. *Causation*: Prostatic obstruction.

Case 48.—J. W., age 68. Complaint: Pain in the right sacro-iliac region coinciding with periods of scanty micturition, also recent pain in the perineum which prevents him bending enough to put on his shoes. Frequent and at times difficult micturition. Prostatectomy had been performed in two stages three years previously. Some months after this he had passed stones intermittently. Urine heavily infected with staphylococci and *B. coli*. One large diverticulum above and external to right ureteral orifice, a smaller one on the left lateral wall, and many shallow sacculi. One large stone in the bladder. Operation: Lithotomy—the calculus weighed 43 g.—and excision of the large diverticulum containing one

calculus weighing 35 g., and three smaller stones. Well for fourteen months, then a return of bladder symptoms, but he remained in fair condition for a further six months, when left pyelitis, and soon after right, developed, and he died of uræmia. *Causation*: Prostatic obstruction.

Case 49.—C. M. G., age 52. Complaint: Difficult micturition for eighteen months. Bladder distended to umbilicus and urine very septic, retention and catheter thrice daily for a fortnight. A stricture treated twenty years ago. Large diverticulum above and behind left ureteral orifice. Operations: Diverticulectomy. After the wound had closed, the residual urine was 12 oz. On re-examination Marion's disease of the col vesicae was recognized. Perurethral resection reduced residual urine to 1 oz. Cured. *Causation*: Stenosis of the col vesicae.

Case 50.—F. J. P., age 60. Complaint: Difficult micturition for four and a half years. A diverticulum recognized at that time, but treatment apparently not advised. One month previously retention of urine; bladder, distended to umbilicus, drained by catheter. Urine very septic. A diverticulum with an unusually large orifice, admitting three fingers, above and external to left ureteral orifice, and a small shallow one more to the right. Cystograms gave appearance of 'hour-glass' bladder. A marked intravesical projection confined entirely to the left lobe of the prostate. Operations: Diverticulectomy; the smaller sacculus was not considered to require treatment. Awaiting prostatectomy. *Causation*: Prostatic obstruction.

Case 51.—L. V., female, age 43. Two years ago acute retention owing to urethral stricture, which was dilated. Three subsequent attacks, the volume of urine being always more than 1 pint. Cystoscopy: A medium-sized diverticulum above and behind left ureteral orifice, with trabeculation and sacculation in the corresponding area on the right. Cystitis, urine very septic. Treatment: Intermittent dilatation. *Causation*: Urethral stricture.

Case 52.—L. J. C., age 58. Complaint: Hæmaturia twice in the last two years. Never any difficulty in micturition. Urine clean. Cystoscopy: A papilloma obscuring the left ureteral orifice and neighbourhood, and extending up to the lip of a small benign diverticulum about 4 cm. deep, situated above and behind the left ureteral orifice. Stenosis of the col vesicae present. Treatment: Diathermy of papilloma. *Causation*: Stenosis of the col vesicae.

Case 53.—D. E., age 58. Complaint: Retention of urine with recent onset. Very uræmic. Examination revealed enlarged prostate. Retention treated by catheterization and cystotomy without exploration. At subsequent prostatectomy a diverticulum the size of a pigeon's egg and one smaller were found immediately posterior to the apex of the bladder. Operations: Cystotomy, diverticulectomy, aud prostatectomy. *Causation*: Prostatic obstruction.

SUMMARY AND CONCLUSIONS

1. Fifty-three cases of vesical diverticula are described.
2. Diverticula are rarely congenital in origin.
3. Their formation is in most cases due to an obstruction to the outflow of urine from the bladder.
4. They are rare in the female, in which sex obstruction is uncommon.
5. Infection and calculus formation are frequent complications; neoplasms are less common.
6. Cure is effected by diverticulectomy, combined with treatment of obstruction if present. A careful search for this must always be made.
7. Diverticulectomy was performed in 36 cases, with 1 death due to the operation.

I am indebted to my late house surgeon, Dr. David A. Lowe, at the Miller General Hospital, for the diagrams *Figs.* 601 and 602, which were made at the time the cases were operated upon. These have been drawn in further detail by Miss M. Leslie Paton, who also made the drawings shown in *Figs.* 593 and 595.

OPERATION SUPPLEMENTED BY OBTURATOR IN THE TREATMENT OF CLEFT PALATE*

By R. R. FITZGERALD, Montreal

Patients with cleft palate fall into two groups. In the first group the gap is narrow, the volume of the available soft tissue is adequate, and when phonation is attempted, the movements of the elements of the palate, lateral pharyngeal walls, and Passavant's cushion are brisk and have good amplitude. Under such favourable conditions it is justifiable to attempt to close the cleft by suture, with the object of securing a palatal valvular mechanism approximating the normal. Although this operation has occupied a place in surgical practice since Langenbeck popularized the lateral relaxing incisions, it is only in the last few years that Veau[11] has established the custom of assessing the results of operative treatment according to the quality of speech later developed. Wardill[15] has also brought forward the functional results in his patients. Pagnamenta[8] has done the same in a less detailed fashion. Now that the practice of publishing speech results has been established, the day is past when it would be possible to consider the appearance of the sutured palate as the criterion of success. If perfect phonation and articulation are secured the operation is a complete success, no matter what the palate may look like. On the contrary, if the speech is defective the operation is a failure, no matter how perfect the palate may be in size, shape, position, or mobility.

To obtain statistics of significance a surgeon is now obliged to submit his patients after operation to examination by an independent observer who is qualified to determine the percentage of patients whose speech is normal, and to classify the remainder according to the standards of modern phonetics. In this way comparable series of cases may be assembled for the purpose of evaluating rival methods of treatment. The achievement of Veau[12] in obtaining normal speech in 62 out of 100 patients has set a high standard for those who would undertake this type of surgery.

In the second group the gap is wide, the volume of available tissue scanty, and movements feeble or absent. These deficiencies may be primarily congenital, or they may result from failure of previous suture operations. In this group any attempt to imitate the normal palatal valvular mechanism by simply joining the soft tissue in the midline is doomed to failure. The reconstructed palate will be so short that it cannot make contact with the posterior pharyngeal wall; the valvular action will be deficient; the speech cannot be normal.

In attempting to treat patients in this second unfavourable group, many have sutured the soft tissues in the midline, producing a short palate, and have then performed secondary operations to complete the cure. Wardill,[14] Padgett,[7] and others have tried to make up the defect in volume of tissue by operations on the posterior pharyngeal wall (pharyngoplasty). Browne[3] has described an encircling

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suture designed to draw the soft palate backward and to approximate the side walls of the pharynx. In the literature there are described many plastic methods of lengthening the palate. Dorrance[4] developed a technique for liberating the short palate and displacing it backward to a new position. The method is to be used only in cases of cleft soft palate. Finally, in those patients in whom the soft tissue is so scanty that there is no prospect of joining the two sides by suture, success has been reported by Fitzgibbon[6] and others by the construction of a metal obturator bearing a hollow chamber on its posterior part. This chamber is gripped by the side walls of the pharynx and effects closure.

Gillies and Fry[1] suggested that the problem in this second group could best be solved by using the available palatal tissues to construct a soft palate, and then closing the anterior opening with an obturator fastened to the teeth by metal clasps.

The present communication deals with 9 examples of this second group of difficult cases, and is a record of an attempt to apply the principles advanced by Gillies and Fry. In five of the nine cases reported there had been previous operations elsewhere, with failure of union. The remaining four were primary cases. In all nine patients plaster-of-Paris models of the palate were made from impressions in dental compound. Study and measurements of these models showed that the gap in each instance was so wide that simple closure by median suture of the soft tissue would produce a palate too short to reach the posterior wall of the pharynx.

MOVEMENTS OF THE PALATE

It has been demonstrated by the studies of Wardill and Whillis,[13] Polland and Hala,[9] and of Borel-Maisonny[2] that there are three elements in the movement of the normal palate in closing the oro-pharynx from the naso-pharynx. There is an upward and backward swing of the soft palate produced by the action of the levators; a forward bulging of the cushion of Passavant by the action of the upper border of the superior constrictor; and a movement of the lateral pharyngeal walls toward the midline (presumably by the sphincter action of the pterygopalatinus, assisted by the salpingo-pharyngeus). Air-tight closure is thus the result of three synchronized movements.

If a case of cleft palate is examined and no movement on phonation is to be seen in the palatal elements, or in the lateral pharyngeal walls, or in Passavant's cushion, it is useless to construct a soft palate. No valve action can possibly be developed, and operation is not warranted. But if the lateral pharyngeal walls move toward the midline on phonation, and Passavant's cushion can be seen to rise on the posterior pharyngeal wall at the same time, one is justified in constructing a soft palate and inserting an obturator in the hope of obtaining good valve action, even though the elements of the soft palate themselves have no voluntary motion. In *Case* 6 of this series the elements of the soft palate were immobile before operation, and three years after successful construction of a soft palate there is still no movement, but the patient has a competent valve and Group 5 (normal) speech. Similar observations have been made by Wardill and Whillis.[13]

Thus it appears that although the upward and backward swinging movement of the soft palate is highly desirable, the movements of the lateral and posterior pharyngeal walls are indispensable. *An adequate valve action can occur through pressure of the sides and back of the pharynx against an immobile soft palate.*

REQUIREMENTS FOR THE OPERATION

The cases suitable for this procedure are those in which there is sufficient soft tissue to build up a transverse bridge across the pharynx, providing that this bridge can be constructed close to the posterior pharyngeal wall, and providing that the lateral and posterior pharyngeal walls move briskly and with amplitude when phonation is attempted. It goes without saying that vigorous voluntary movements of the soft palatal bridge are also highly desirable, and that the efficiency of the valve increases with the strength and amplitude of such movements.

It would be wise to warn against expecting success from this operation if the general health, and especially if the mentality, of the patient is below normal. In the cases here reported the intelligence quotient (I.Q.) was estimated in each case where the child was old enough, and no child with an I.Q. of less than 50 was accepted for the operation. The speech results linked with I.Q. in *Table I* are of some interest. While we have found that the children with a high I.Q. can learn quickly, there are a number with quite low standards of intelligence (I.Q. 53–67) who have almost normal speech (Group 4) as a result of patient and prolonged speech training. On the other hand, there is a group of 3 with I.Q. between 80 and 90 who still have inferior speech (Group 2). The reason for refusing to operate on children with an I.Q. below 50 is that success cannot be obtained in the absence of ambition, capacity for learning, or a desire to attain correctness in articulation.

The services of an expert speech trainer are indispensable to establish and maintain new and correct habits in articulation and phonation. In this series there are instances in which two years of painstaking instruction in the speech-training centre, together with co-operation of intelligent parents and teachers outside the hospital, have been necessary before results began to be apparent in overcoming the vicious speech defects acquired before operation.

Table I.—Showing Speech Results Obtained in Cases with Varying Intelligence Quotients

Operation	Case No.	Age	Intelligence Quotient	Pharyngoplasty* (Wardill)	Speech Group† (Scharfe)
Gillies and Fry	1	13	94	+	3
	2	12	82	o	5
	3	3	Too young to test	o	Too young to test
	4	4	Too young to test	+	Too young to test
	5	9	56·5	o	4, *b*
	6	11	70	o	5
	7	9	81	+	4, *c*
	8	9	53	+	4, *b*
	9	16	86	+ +	2, *c*
Short palate modification	10	20	67	+ +	4, *b*
	11	17	83·5	+	2, *d*
	12	13	102	+ +	5
	13	11	57·5	+	2, *b*

* +, pharyngoplasty performed; + +, pharyngoplasty performed and carried out a second time o, no pharyngoplasty.

† The speech groups are those referred to in *Table II.*

Decayed teeth and diseased tonsils should be removed. Anæmia, syphilis, or chronic sinus disease will spoil the result, and must be treated beforehand. Finally, the most important part of the operation is the constructing, fitting, adjusting, and care of the obturator, and unless the help of a sympathetic dentist specially skilled in prosthetic work is constantly available, there can be no prospect of success.

ANÆSTHESIA

Avertin as a basal anæsthetic, supplemented by gas-oxygen by the endotracheal method of Magill, has been used with satisfaction in this series.[5] The endotracheal tube has been passed through the nostril, except in those cases where Wardill's pharyngoplasty[14] has been a part of the operation. In such cases the tube is passed through the mouth.

THE OBTURATOR

A cast of the palate is made in plaster-of-Paris from an impression in dental compound, and the dimensions of the proposed obturator decided upon by measurement of the model and of the mouth. The completed plate has to be imagined, and the obturator designed in wax. Retention is obtained by adaptation to the overhang of the sides of the hard palate cleft. Gold clasps adjusted to the teeth are also used if suitable teeth are present. If the teeth are peg-shaped, or are not placed at a favourable inclination for the clasps, we do not hesitate to insert a small metal pin in a drill hole on the labial surface of a tooth to hold the clasp in place. The obturator is then made of vulcanite. In edentulous patients the upper part is made of soft rubber to be gripped by the bony margin of the cleft, and the buccal plate constructed in vulcanite. The posterior edge of the obturator is grooved to receive the anterior edge of the new soft palate. The completed obturator thus has an anterior part, designed to fill whatever defect there may be in the hard palate area, and a posterior part, which occupies a portion of the space normally filled by the soft palate. We have had no success in our attempts to insert a hinge between the two parts. There is always an angle between the anterior and posterior part to imitate the difference in inclination between the hard and soft palates in the normal. Only experience can tell just what this angle should be. In some instances it has approached a right angle; in others the two parts had to be constructed on almost the same plane.

OPERATIVE TECHNIQUE

With the child in the Rose position and a Dott mouth-gag in place, the mouth is cleaned with 1–150 carbolic acid solution and the pharynx packed with gauze to prevent swallowing of blood. The endotracheal tube itself protects the lungs. The steps of the Gillies and Fry operation are illustrated in *Fig.* 603.

The posterior pharyngeal wall is first examined, and a decision reached as to whether or not pharyngoplasty should be carried out. This step, when performed by the method described by Wardill,[14] is quick, and I believe, safe. It has been employed wherever the pharynx has been wide. In three patients the operation has been done a second time in an attempt to obtain the maximum improvement.

It was used in five of the patients without accident, and without destroying the cushion of Passavant. In *Table I* the patients in whom pharyngoplasty was performed are indicated in the fifth column, and the speech results in the sixth. When the intelligence quotient of some patients (column 4) is considered, it seems to be proven that in this series pharyngoplasty has had a favourable influence on the final speech result.

After the pharyngoplasty has been completed a Reverdin needle is inserted, according to the method of Veau,[11] into the soft palate close to the pharyngeal wall on the patient's right side. The needle is passed through the mucosa and

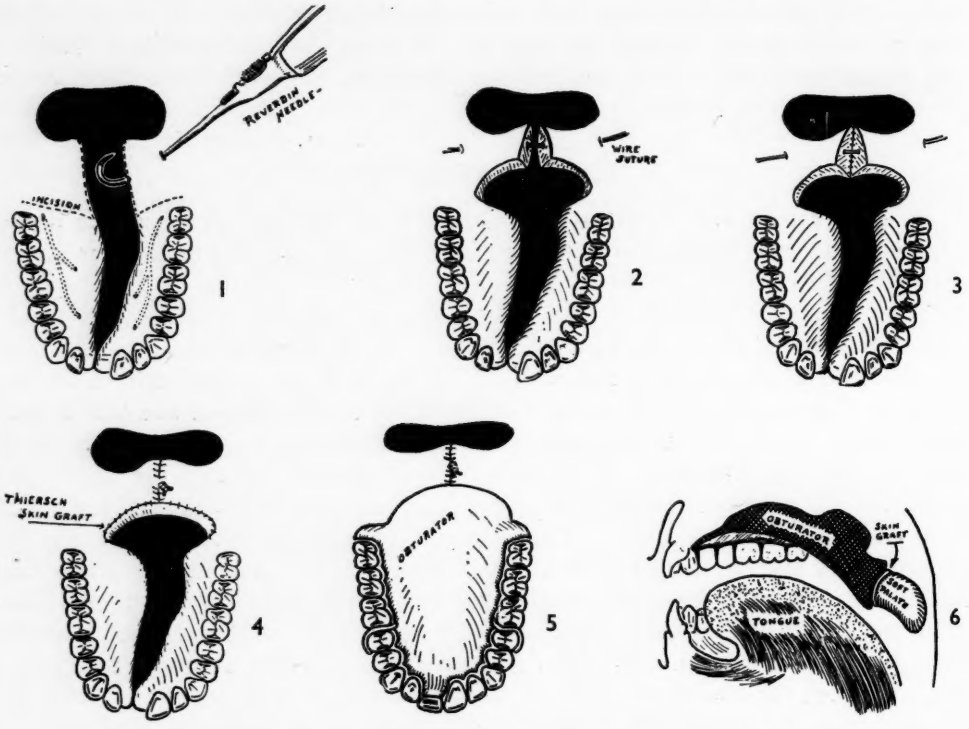


FIG. 603.—Details of the Gillies and Fry operation. 1, Placing Reverdin needle; incisions. 2, Wire suture in position. 3, Nasal layer sutured. 4, Wire suture completed; buccal layer sutured; skin-graft sutured in place. 5, Obturator in place. 6, Section to show position of obturator, skin-graft, and soft palate.

muscle, and is then made to pass toward the midline *in the nasal submucous layer*. The point of the needle is pushed through the mucous membrane in the edge of the cleft, and, using the needle to steady the tissues, the margin is split backward to the uvula and forward to the hard palate, creating a nasal and a buccal free edge of mucous membrane. The soft palate is then freely separated from the hard by scalpel and scissors. The separation is carried just far enough laterally to allow the soft tissues to swing backward and medially to the desired position. The pterygoid hamulus is generally seen, and a branch of the descending palatine artery may be divided. A bronze wire suture 0·1 mm. in diameter is threaded, and the needle withdrawn. The manœuvre is repeated on the patient's left side, and the wire suture drawn through.

Suture of the nasal mucosa is now begun at the anterior end, using No. 0 chromicized catgut, and tying the knots on the nasal side. The uvula is reconstructed, and suture of the buccal mucosa begun, from behind forward.

Interrupted dermol has been used for suture material. When the buccal mucosa has been closed as far forward as the wire, the muscle suture is completed and the wire tied and twisted according to the instructions contained in the writings of Veau.[11] A few more sutures of dermol complete the closure of the buccal mucous membrane. The new soft palate has a thick, raw anterior edge, and the angles on each side of it are also uncovered by epithelium. A Thiersch epidermal split skin-graft is next cut from the medial surface of the patient's arm, and sutured in place with fine dermol on the anterior edge of the newly formed soft palate; it is also carefully adjusted to cover the raw surfaces where the soft palate has been separated from the lateral pharyngeal walls. It is not necessary to cover the bare bone of the posterior edge of the hard palate with the skin-graft.

The obturator is now fitted in place, and securely fixed by fastening one or more of the clasps to the teeth with stainless steel wire. Its grooved posterior edge must hold the skin-graft firmly against the anterior border of the new soft palate. The free posterior edge of the soft palate should be in such a position that it can be applied against the posterior pharyngeal wall by a light touch.

POST-OPERATIVE CARE

The child's elbows are splinted to keep the fingers from the mouth. Food is offered as soon as there is prospect of its being retained, and every effort made to keep the patient from crying. Paragoric is given freely as a sedative. The obturator is not removed for one week, in order to allow the skin-graft to take. It is then removed daily for cleansing. The soft palate constructed in this way has a strong tendency to creep forward. If the obturator is left out for a few hours it cannot be replaced without using some force under an anæsthetic. This tendency to creep forward persists for years. If the child loses or breaks the obturator and has to go a few days without it, the soft palate immediately begins to move forward to an anterior position.

RESULTS

From an anatomical standpoint there have been no failures. The two sides have always joined by primary union, and the skin-graft has always survived. The children wear the obturators without discomfort; in fact they always refuse to have them out for any length of time once they have been satisfactorily fitted. Much more interest has centred around the functional than the anatomical result. My colleague, Dr. E. E. Scharfe, Director of the Speech Training Clinic at the Children's Memorial Hospital, has kindly examined my patients before operation and has graded them according to the scheme which he has described[10] for classifying cleft-palate speech.

The patients have also been re-examined by Dr. Scharfe at the present stage of their treatment, some completed, some still under training. The actual effects on speech that can be credited to the operation plus careful subsequent speech training are presented in *Fig.* 604.

Speech Group before Operation	Palate before Operation	Palate after Operation	The Obturator	Obturator in Place	Speech Group after Operation	Result
1					3	Improved
2, *c*					5	Normal speech
Too young to test					Too young to test	
Too young to test					Too young to test	Promising
1					4, *b*	Much improved Speech almost normal
1					5	Normal speech
2					4, *c*	Much improved Speech almost normal
1					4, *b*	Much improved Speech almost normal
2, *b*, *c*					2, *c*	Very slight improvement Diminution in the escape of air through the nose

FIG. 604.—Structural and functional results of the Gillies and Fry operation. The speech groups are those introduced by Dr. Scharfe (*see Table II*).

Table II.—CLEFT-PALATE SPEECH (DR. SCHARFE'S GROUPING[10])

GROUP	CHARACTERISTICS
1	Escape of air nasally and failure to articulate consonants (except M, N, and NG)
2	Escape of air nasally and defects of articulation :— *a.* Omission of some consonants *b.* Incorrect articulation *c.* Substitutions (T and D for K and G; W for R; etc.) *d.* Compensatory speech habits :— Glottal stop Pharyngeal and nasal S Constriction of nares, etc.
3	Only escape of air through nose (normal articulation)
4	Defects of articulation only (no escape of air nasally) :— *a.* Omission of some consonants *b.* Incorrect articulation of some consonants *c.* Substitution (T and D for K and G; W for R; etc.) *d.* Compensatory speech habits
5	Normal speech

Perfect speech has been achieved by two patients. Three have slight defects in articulation that can be detected only by experts in phonetics; to the casual ear they have normal speech. One has useful but slightly defective speech. Two promising cases are too young to test. In the one remaining case there has been a slight improvement. Treatment is being persisted in as long as articulation is below normal. It has been learned that the appearance of the completed palate mechanism is no guide to its utility. There have been disappointments, as well as happy surprises.

APPLICATION OF THE METHOD TO CASES OF SHORT SOFT PALATE

In a series of 6 cases in which operation for congenital cleft has resulted in a soft palate too short to reach the posterior pharyngeal wall, the soft palate has been deliberately separated from the hard by operation and displaced backward and downward until it made contact with the posterior pharyngeal wall. A Thiersch skin-graft has been sutured to the anterior edge of the soft palate and an obturator inserted. In all cases this operation has resulted in temporary loss of power of movement of the soft palate, with recovery after a period of months. The method has been used only in patients having active movements of Passavant's cushion and of the lateral pharyngeal walls.

RESULTS

These cases have also been classified by Dr. Scharfe according to the quality of speech before and after operation. In one patient the articulation and phonation are now normal. Three others are improved. Two of the patients are so young

that an accurate analysis of the powers of speech cannot be made. The structural and functional results in the four older children are shown in *Fig.* 605. The

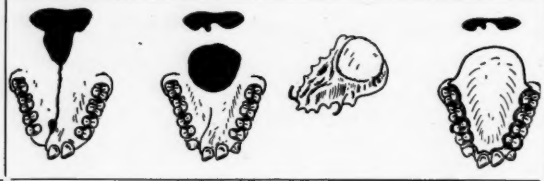
Speech Group before Operation	Palate before Operation	Palate after Operation	The Obturator	Obturator in Place	Present Speech Group	Result
1					4, *b*	Much improved Speech almost normal
2, *c*					2, *d*	Very slight improvement Diminution in the escape of air through the nose
2, *d*					5	Normal speech
1					2, *b*	Slight improvement

Fig. 605.—Structural and functional results in 4 cases of short palate treated by insertion of an obturator after separating the soft from the hard palate and suturing a skin-graft to the raw edge. The speech groups are those described by Dr. Scharfe (*see Table II*).

younger patients seem to derive more benefit than the older ones, in whom our efforts in speech training seem to be wasted. It is possible by this operation to construct a palate mechanism that can function as an adequate valve under the orthodox tests (snorting, blowing balloons, stethescope test for escape of air nasally, etc.), but making a patient 'valve conscious' is another matter. A patient of intelligence with a good ear for tone may pick up the knack of using the valve in a few weeks. Others do not seem to be capable of grasping the idea of using the muscles to prevent the sounds from escaping through the nose.

SUMMARY

In 9 cases (4 primary, 5 secondary) of congenital cleft palate with wide gap and scanty soft tissue, the method of Gillies and Fry has been used, and has resulted in improvement in speech in all of the patients who are old enough to undergo tests. A similar method has also been applied to 6 cases of post-operative short palate, with improvement of the speech in 4 instances.

It is a pleasure to acknowledge the assistance of Dr. E. E. Scharfe and Mrs. Ismay, of the Speech Training Clinic, Dr. R. W. Wheatley and staff of the Department of Dentistry, and Dr. T. Ivan Guilboard, who has been responsible for much of the technical dentistry, and who has drawn the figures illustrating the paper.

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THE CLINICAL COURSE AND PATHOLOGY OF BURNS AND SCALDS UNDER MODERN METHODS OF TREATMENT*

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In 1933 a short report on the treatment of men burned in colliery explosions[23] was rendered to the Ministry of Mines by a committee under the Chairmanship of Sir David Wilkie. The report, though concerned mainly with measures to lessen the high death-rate from burns sustained in colliery explosions, contained also some details of an investigation into the causes of death in burns. The observations reported therein were at variance with views which had been expressed in recent literature on the subject, and, as these views have subsequently gained new adherents, it seemed desirable that the work initiated by the committee should be continued and extended. The present paper is a record of the continued investigation into the causes of the systemic disturbances and of death after burns under the conditions of modern methods of treatment. Our study of the clinical course has, it is hoped, provided a fuller description and a clearer definition of its stages than has hitherto been available; in the section on the pathology of burns, consideration has been given to aspects not previously emphasized. We have recorded our conclusions as to the causes of death after burns, and made some reference to treatment and its future developments.

THEORETICAL CONSIDERATIONS

In the following short survey of theoretical aspects, we shall refer only to literature which deals particularly with the causes of the systemic disturbances and of death after burns. A more comprehensive account of the subject may be found elsewhere.[2, 9, 19, 21, 29] It is customary and convenient to discuss the clinical course in respect of stages because at successive periods after injury the systemic disturbances differ in nature and probably in causation. For the purpose of the present discussion we shall employ a classification in common use, whereby the clinical course is divided into four stages. The first is *shock*, which occupies a variable period during the first twenty-four hours; the second is *toxæmia*, which may continue till the 4th or 5th day; the third is *sepsis* or bacterial intoxication; and the fourth is *healing*. In this section we shall mention only the aetiology of each stage.

1. **Shock.**—Shock which follows injury by burning so closely resembles shock following severe trauma that there seems no reason to doubt that aetiologically the conditions are essentially similar. The aetiology, however, is not yet fully determined.

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The important work of the Medical Research Committee[25] which investigated surgical shock and allied conditions during the late war, is too well known to require detailed recapitulation at this time. Shock was divided into 'primary' and 'secondary' shock; the first was attributed to derangement of the vasomotor mechanism by afferent nerve-impulses; the second was thought to be probably due to the action of a histamine-like depressor substance which had been absorbed from the injured tissues—that is, secondary shock was considered to be of the nature of a toxæmia. The theory of the nervous origin of primary shock has not been seriously questioned. That of the toxic origin of secondary shock has, however, become scarcely tenable in view of subsequent investigation. In large part the theory rested on experiments by Cannon and Bayliss[7] in which shock was produced by trauma to muscles. Among subsequent investigators there has been general agreement that Cannon and Bayliss, in suggesting that absorption of toxins was the probable mechanism of shock production, had underestimated the effects of local loss of blood or loss of blood and plasma combined.[20, 3, 15] According to O'Shaughnessy and Slome[18] they had failed also to exclude properly the influence of afferent nerve-impulses. Experiments devised to test for depressor substances in blood from a traumatized limb have also given negative results.

At present three alternative explanations, two of which are revivals of older theories, obtain greater favour. The first is that secondary shock is due mainly to loss of blood or plasma, or both, at the site of injury. The second is that it is due mainly to nociceptive nerve-impulses, and that local escape of fluid into the damaged tissues is a subsidiary factor. The third, which has been suggested by Cannon[6] on the basis of experiments by Freeman,[12] is that hyperactivity of the 'sympatho-adrenal system' plays at least a contributory part. Those who hold that secondary shock is essentially of nervous origin naturally dispense with a classification into primary and secondary shock.

Needless to say, each theory has been suggested as an adequate explanation for shock following burns, although for only one has any evidence been offered in support; Blalock[4] and also Harkins[13] concluded that the local loss of plasma in the burned area was sufficient to produce a pronounced fall in blood volume, and that it was probably the chief factor in the causation of shock.

It would be outside the scope of this article to criticize these theories or to detail the evidence for and against each. It seems to us that the possibility of a toxic factor in the causation of shock in man has not yet been entirely excluded.

2. Toxæmia.—The next stage, that of 'toxæmia', has been at least appropriately named, because in its fully-developed form the condition resembles an overwhelming poisoning. Three mechanisms have been suggested as the cause of the systemic disturbances:—

a. The Action of Toxins which have been formed in the burned area and absorbed into the circulation; the evidence for this mechanism has been reviewed previously.[2, 9, 21, 29, 33]

b. Anhydræmia or Increased Concentration of the Blood.—Underhill[26, 27, 28] has been the most active proponent of this view, according to which large quantities of fluid escape from the blood into the burned area, and in consequence the blood concentration and viscosity increase to such a degree as to impair circulatory efficiency. The importance of anhydræmia as a cause of systemic disturbances has been much stressed in recent years,[16] and it is commonly believed that

enormous quantities of fluid can be lost from the circulation into an extensively burned area.

Other points in Underhill's thesis are that absorption from burned tissues is greatly reduced, that chlorides as well as water are retained in the burned area, and that the blood-chlorides are depleted. To the combination of fluid and chloride depletion of the blood Underhill has attributed all or most of the systemic disturbances of burns during the first few days, and he has rejected emphatically the possibility of toxin formation or absorption from burned tissues. There is also evidence that in man the blood-chlorides may diminish and other changes in blood-chemistry occur.[10, 11]

It may be stated, however, that clinical experience in general does not support Underhill's contentions, because fluid administration in itself will not prevent toxæmia and death,[1] and, under certain conditions of treatment, fatal toxæmia can occur without evidence of serious anhydræmia.[23, 30] Still more decisive evidence is contained in this paper.

c. Bacterial Infection of the Burned Area.—Aldrich,[1] who was as sceptical as Underhill of a specific burn toxin, substituted the hæmolytic streptococcus for anhydræmia as the chief causal agent. His further claim that the application of gentian violet instead of tannic acid to the burned area greatly diminished the incidence of streptococcal infection has not been universally accepted.[24, 16]

The present trend of opinion seems to be towards Underhill's view that increased concentration of the blood is the main, if not the sole, cause of symptoms in the stage of toxæmia; some support,[8] however, is given to Aldrich's belief in the predominating influence of hæmolytic streptococcal infection. An attitude of scepticism or reserve towards a specific burn toxin has become fashionable.

3. Sepsis.—That bacterial invasion of the burned area is of frequent occurrence and a source of great danger is well-known, and that the hæmolytic streptococcus is the most usual agent of serious infection is a common experience. The only points in dispute are the time of onset and the frequency of serious infection. Aldrich stated that streptococcal infection was common and that it occurred early, often within twenty-four hours. The usual estimate has placed the onset towards the end of the first week.

4. Healing.—This stage requires no discussion here.

METHOD OF INVESTIGATION

Our investigation was directly towards the points which, as indicated in the foregoing section, were most in dispute. As will be seen, it has yielded information mainly in relation to the causation of toxæmia and the incidence and time of onset of bacterial infection.

From more than 200 cases of burns which came under our care or were specially referred to us for the purposes of the investigation, we selected for study 65 cases in which the injuries, by reason of their extent and severity, seemed likely to prove serious. The observations were therefore concerned almost exclusively with severe and dangerous injuries, and the majority, 57 of 65, were in children below 12 years of age. In 41 instances the injuries were scalds, in 24 burns by fire. Twenty-three (15 scalds and 8 burns) of the 65 patients died, and post-mortem examination was obtained in 20. Additional pathological material was obtained from 13 other

fatal cases, in most of which the clinical course was observed by us, but circumstances did not permit of special clinical investigation.

Observations were made on blood-pressure, heart-rate, respiration-rate, rectal temperature, and blood and urinary changes. Blood examination included concentration, as estimated by the hæmoglobinometer (Haldane), leucocyte counts, sedimentation rate (Landau's micro-sedimentation method), bacterial cultures, and chemistry. The usual blood-chemistry investigation comprised chlorides, carbon-dioxide combining power, and plasma proteins; in some cases additional estimates were made, such as non-protein nitrogen, urea, cholesterol, inorganic phosphorus, serum calcium, potassium, sodium, van den Bergh reaction, and icteric index. The burned areas were examined at intervals for bacterial growth by anaerobic and aerobic cultures. Cultures were taken from small incisions made through previously sterilized portions of the coagulum. In a few cases gastric acidity was measured.

The post-mortem examinations included the brain, thyroid, parathyroid glands, lymph-glands, burned and unburned skin, and all thoracic and abdominal organs; bacteriological cultures from blood and burned areas were made.

As the investigation proceeded, its scope was extended, and thus the studies were more complete in later than in earlier cases of the series. In addition to the selected cases, many others were investigated for a limited period, till it was clear that they would yield no information of special value.

The main principles and details of treatment may be described here; treatment was the same for all cases unless otherwise stated in case reports. Immediately the patient was admitted to hospital, morphine or heroin was administered, and as soon as practicable the local treatment of the burned area was carried out under nitrous oxide, oxygen, and ether anæsthesia. All loosened epidermis was removed by gauze soaked in warm normal saline solution, and to the raw surface 20 per cent tannic acid with the addition of 1 per cent gentian violet (in some cases 1–1000 acriflavine) was applied. Two applications of the coagulating solution were made, and the area was dried by a current of hot air from a hair-drier after each application. Immediate coagulation of the surface layer of the burned area was thus produced. Thereafter the area was exposed to the air under a heating cage (*see Fig.* 618) and supervision was carried out as previously described.[23, 31] In many cases intravenous infusions were given during the early stages; these and other adjuvant measures are mentioned in individual case reports and in the discussion on treatment.

To present the results in the most convenient form, we shall give first a general description of the clinical course and of the pathology of burns as they were exemplified by our cases, and later we shall detail the evidence for our conclusions by quoting illustrative cases.

CLINICAL COURSE

The severity of the constitutional disturbances which followed injury by burning was influenced by many factors, of which the most important were the age of the victim, the extent of skin surface involved, the depth and situation of the burn, and the time which elapsed between the infliction of injury and the institution of treatment. In any group of cases, however, in which these and other factors were fairly constant, very conspicuous differences in the clinical course were observed. There seemed little doubt that the main cause of such differences was

individual variation in susceptibility to the effects of injury. In a proportion of the cases selected for study the constitutional disturbances were very mild, or even absent. In other instances, however, they were more or less pronounced, and followed a course which may be regarded as characteristic of this form of injury. In representing the course we have adhered to a classification, previously employed by one of us,[30] which differs slightly from the usual classification, and in which the course is divided into *five* stages: (1) Initial shock; (2) Secondary shock; (3) Acute toxæmia; (4) Septic toxæmia; and (5) Healing. It should be noted that there are wide variations, in any group of comparable cases, in respect of the severity and duration of each stage. The classification has been employed because it is useful for description of the clinical course, and because it seems to give, on the whole, the most accurate representation of the systemic disturbances.

1. INITIAL SHOCK

We have restricted the diagnosis of initial shock to the condition of low blood-pressure in cases which were admitted to hospital within 2 hours of injury. The criterion of blood-pressure level was essential for satisfactory diagnosis and analysis; the period of 2 hours, though arbitrarily chosen, proved convenient for purposes of classification.

Forty cases of the series were admitted within 2 hours of injury; in 35, and amongst these were some of the most extensive and severe injuries, the systolic level of blood-pressure was normal or, in a few, actually above normal. That is, initial shock was present on admission in 5 cases only; in 2 it was severe, in 3 moderate or mild in degree.

The 2 cases of severe initial shock were associated with unusual circumstances. Unfortunately in neither was investigation sufficiently complete to provide satisfactory information. One was complicated by œdema of the glottis and pronounced anoxæmia; following tracheotomy the blood-pressure regained a normal level, but the circulation failed later and death occurred at 21 hours. The other had very extensive and deep burns; initial shock, which was profound on admission at 1¼ hours, gradually deepened till death at 7 hours. Here there was evidence, from post-mortem examination, of unusual pathological features, namely infiltration of the liver and spleen by eosinophil leucocytes, which will be referred to again. In the cases of moderate or mild initial shock the maximum fall of systolic blood-pressure was 24 mm. Hg; the other clinical features were pallid cold skin, sub-normal rectal temperature, feeble radial pulse, which was usually little altered in rate, and apathy or anxiety. Recovery to a normal pressure level occurred within an hour of administration of sedatives and warmth.

In addition to the cases classified as initial shock, there were several which, but for the necessary criterion of hypotension, would have been included as examples of initial shock. Although the systolic level and pulse pressure were unaltered, the condition otherwise was indistinguishable from initial shock, and was similarly responsive to treatment.

During the first 2 hours, and irrespective of the presence of shock, there was no noteworthy change in blood-chemistry or corpuscular sedimentation-rate. The hæmoglobin content of venous and capillary blood was also usually normal; exceptions are mentioned in the description of secondary shock. A leucocytosis in venous and capillary blood was a frequent, though not invariable, occurrence. The count

was sometimes surprisingly high; 38,000 per cmm. at 45 minutes after injury, and 45,000 at 1 hour were recorded. The proportions of leucocytes were unchanged, and in no instance were eosinophils in excess. The leucocytosis was not merely a consequence of increased concentration, because in nearly all instances of high counts the hæmoglobin content was normal. The mechanism has not yet been investigated. A number of agencies, such as reflex nervous action or increased secretion of adrenaline, which has been found after experimental burns,[14] might be suspected.

Even in this series of severe injuries, therefore, initial shock was infrequent, usually of moderate severity, and transient. Profound initial shock was associated with unusual circumstances.

2. SECONDARY SHOCK

As examples of secondary shock we have included cases in which shock developed subsequent to admission and also those in which hypotension was already present at admission later than 2 hours after injury; in the majority of the second group the interval between injury and admission was considerably greater than 2 hours.

Development.—In most instances of secondary shock in the present series, the condition began during, or immediately after, local treatment, and the development was then rapid. The development of secondary shock before local treatment was usually gradual, though 2 cases of rapid development were observed. In cases of gradual development of shock, the pulse pressure decreased steadily over a period of 1 or 2 hours, the systolic level falling slightly while the diastolic level rose; after this period both levels fell rapidly and on occasion the fall was sudden and pronounced. In severe shock the systolic pressure fell below 60 mm. Hg. (adults), and we have in fact observed systolic levels of 25 and even 16 mm. Hg. Simultaneously the quality of the radial pulse deteriorated and the heart-rate increased.

Established Shock.—The well-known features of surgical shock always appeared once the main collapse of arterial pressure had begun; sometimes they preceded it by many minutes. In severe shock the chief complaint was thirst, which was often almost insupportable; pain, on the other hand, was rarely prominent unless the hands or face were injured. Adults were usually alert and distinctly anxious, while children tended to be apathetic except for the constant and intense desire to assuage thirst. The skin was cold, pale, and clammy; the lips, cheeks, and ears were ashen-grey in colour (the greyness being accentuated sometimes by impregnation of the skin with carbon particles); the rectal temperature was subnormal; respiration typically was shallow and rapid, though in the most profound shock the irregular exaggerated movements of 'air-hunger' appeared, together with great restlessness and distress. Vomiting was practically invariable; fluid drunk to relieve thirst was always vomited very shortly afterwards. Capillary blood from ear or finger-tip usually showed a distinct rise in hæmoglobin content; an average figure was 115 per cent and a rise to 150 per cent has been found. Occasionally venous blood showed a similar rise, though usually of lesser degree, but more often the venous blood hæmoglobin was normal. No constant change in blood-chemistry was observed, and even the carbon-dioxide combining power, a fall of which is generally regarded as characteristic of traumatic secondary shock, was rarely significantly lowered. In one recent case, however, a distinct fall in serum sodium has been recorded—a feature requiring further investigation.

In moderate or mild degrees of secondary shock the changes were correspondingly less pronounced; the systolic level remained above 60 mm. Hg, there was little change in concentration of capillary or venous blood, and no alteration in blood-chemistry.

Incidence and Consequences.—The incidence and degree of secondary shock were closely related to the extent of skin surface which had been injured; the greater the extent, the more rapid and severe the shock. Extent was not the only factor, however. In most, though not all, cases in which secondary shock became severe, the burns were deep as well as extensive. As already mentioned, the procedure of local treatment appeared to precipitate secondary shock, or to aggravate it if already present. Usually signs of shock were inconspicuous before local treatment, but if more than 20 per cent of the skin surface were injured, some degree of secondary shock invariably developed during or after the procedure. Treatment, of course, introduced factors liable to aggravate shock, such as anæsthesia, the production of painful nerve impulses, and some additional loss of plasma from the injured surface. Nevertheless the regularity of the development of secondary shock, even when every effort was made to mitigate the effect of these factors, was striking. It was possible also to demonstrate the specially deleterious actions during shock of open ether anæsthesia, of anoxæmia, and of the application to the injured surface of irritant cleansing agents such as ether and alcohol.

The capacity for spontaneous recovery was present, though uncertain, in moderate and mild degrees of secondary shock. On the other hand, in severe degrees the circulatory collapse was continuous and progressive, and the course, we are convinced, judging from our own experience and that of others, would invariably have terminated fatally in 12 to 24 hours after injury but for the active therapeutic measures employed. It may be noted that no patient treated by the methods previously outlined died from secondary shock.

3. ACUTE TOXÆMIA

Acute toxæmia, even in this selected series of dangerous burning injuries, was frequently absent, or was so mild as to amount to nothing more than an inconsiderable and unimportant malaise. In a number, however, it occurred in severe form, and at its worst it resembled the overwhelming intoxication produced by the most virulent bacteria. This fulminating form, which was most frequently observed in young children and infants, had very constant and characteristic features, which will now be described.

Severe Acute Toxæmia in Children.—The onset was as a rule insidious and the development gradual, and even when the condition appeared to make a late and abrupt entry, there were premonitory signs that all was not well. The onset was at any time between 6 and 50 hours after injury, in the majority at about 12 to 15 hours, and the first sign was usually *vomiting*, which was more continuous and persistent than could readily be attributed to the preceding anæsthetic. Altered blood in the vomit at an early stage presaged a serious illness. Simultaneously a highly characteristic disturbance of the *mental state* began. The child became apathetic and listless, and yet curiously, and sometimes intensely, irritable and restless, made repeated efforts every few seconds to change position, and evinced special resentment at finding the movement of the limbs restrained. Drowsiness was evident, yet sleep was fitful and was frequently interrupted by jerking movements

of limbs and trunk. The restlessness was very little influenced by ordinary doses of sedatives, and gradually merged into delirium, stupor, and finally coma. In two infants recurring epileptiform convulsions preceded coma, which deepened after each convulsion. The mental disturbances usually appeared before any serious failure of the circulation, and evidently some process was peculiarly noxious to the brain cells at this stage. Further evidence may be cited here. During the period of investigation, 6 children with small burns were admitted to hospital, between 14 and 50 hours after injury, solely on account of severe cerebral disturbance which overshadowed the other signs of acute toxæmia; 4 had recurring epileptiform convulsions and 2 wild delirium.

Hyperpyrexia.—This was a characteristic feature, and as a rule was persistent and progressive. It may be noted that the hyperpyrexia of acute toxæmia has been considerably modified in our series in which treatment by exposure of extensive skin surfaces has facilitated regulation of body temperature.

Circulatory Failure.—This was responsible for some of the most distinctive signs of acute toxæmia. The radial pulse became progressively more rapid and feeble. The skin of the trunk and limbs became pale, or mottled, and cold; pallor replaced the reactionary erythema surrounding the burned area, thus throwing into sharp relief the dark colour of the tanned surface, while the low temperature of the skin, particularly of nose, ears, fingers, and toes, was strikingly at variance with the high rectal temperature. The greyish-cyanotic hue of lips, cheeks, and ears, indicating stasis in the minute vessels, was further evidence of failure of the peripheral circulation, and at an advanced stage the eyes became sunken and dark-ringed and the pupils dilated. The blood-pressure changes were variable. Usually the systolic pressure remained at or near a normal level till within a few hours of death, while the pulse pressure was diminished by a rise of diastolic pressure; the diastolic level was often, however, very erratic. Less frequently there was a very slow and almost imperceptible decline in systolic and diastolic pressures, terminating in an abrupt fall as the circulation finally failed. In a few cases there were sudden falls in pressure at intervals, associated with signs of collapse. In the early stages recovery to a normal level occurred spontaneously or after intravenous infusion of a colloidal solution, such as gum saline or blood, but the response became steadily more evanescent and incomplete, and usually failed entirely at any time after 60 hours.

Respiration.—In the absence of pulmonary complications respiration was not specially affected. It was not as a rule shallow, and the rate was increased only in proportion to the degree of pyrexia.

During the earlier part of the investigation acute toxæmia of such severe degrees always pursued an inexorable course to a fatal termination between 50 and 100 hours after injury; the average time of death was 70 hours. Recent experience, however, has suggested[32] that in a proportion of cases of severe toxæmia circulatory failure may be successfully treated and the course considerably modified.

Blood Changes.—Even in the most severe and fulminating acute toxæmia there was no constant change in blood concentration. Frequently the hæmoglobin content of capillary blood remained unaltered, and it was unusual to find a significant rise in venous blood. In burns of more than moderate extent there was a tendency for the capillary blood to be concentrated during the first two or three days, but estimations in cases with and without toxic signs elicited no evidence of

any constant relation between increased blood concentration and acute toxæmia. These points are well illustrated in the cases quoted later.

The leucocytosis which often appeared immediately after injury persisted with a steadily diminishing count during the first four days and was unaffected by the advent of toxæmia. During fulminating toxæmia the leucocyte count might remain normal and the rate of corpuscular sedimentation unchanged.

Common changes in blood-chemistry were a diminution in chlorides, carbon-dioxide combining power, and plasma albumin, and a rise in non-protein and urea nitrogen. Such changes, however, were neither constant nor proportional to the severity of the systemic disturbances. Recent investigations suggest that serum sodium and potassium may prove a more reliable index of toxæmia, but only a few observations are at present available.

Urine.—Abnormal constituents were variable; acetone, albumin, and casts were occasional, and bile has been found as early as 48 hours.

Bacteriology.—The cultures, aerobic and anaerobic, of the burned tissues were taken from several points which seemed likely to be reasonably representative of the burned area. Cultures were also made from blisters, fissures, and any raw surfaces which might appear during the first few days. The latter contained various organisms such as streptococci, hæmolytic or non-hæmolytic, staphylococci, and coliform bacilli, but the cultures of 'representative' areas very frequently showed no growth up to 90 hours. If we accept the latter as a true indication of bacterial activity in the burned area, the evidence was against bacterial action as the cause of acute toxæmia. As it happened this evidence was most decisive in cases of severe and fatal toxæmia. Certainly there was rarely any suggestion that hæmolytic streptococci were flourishing in the burned area and invading the blood-stream. Moreover, hæmolytic streptococci were sometimes grown in pure culture from isolated portions when systemic disturbances were entirely absent. If the remarkably severe and rapidly fatal illness were to be explained by bacterial action, we should have suspected not the hæmolytic streptococcus, but rather an organism capable of producing the most potent exotoxin, and probably growing anaerobically under the tanned layer. The local condition of the burned area during life and after death, and the results of cultures, gave not the slightest indication of such a process.

Milder Degrees of Acute Toxæmia in Children.—The features of milder degrees of acute toxæmia were occasional vomiting, slight cyanosis, and a moderate rise of pulse-rate and rectal temperature. They appeared at the usual time and subsided at 30 to 40 hours after injury. One sequel, however, was of special significance, namely *jaundice*, which was observed in 5 such cases. The results of examination of blood concentration, blood-chemistry, and bacterial growth were substantially the same as in the severe grade of toxæmia.

In 3 cases a variation in the clinical course was recorded. Acute toxæmia began at about 12 hours and remained mild, in 2 cases so mild as almost to escape detection, until between 40 and 60 hours, when suddenly it became severe and proved rapidly fatal.

Acute Toxæmia in Adults.—The condition in adults was on the whole less dramatic and distinctive than in children, and survival was more prolonged. The illness was less overwhelming, though several cases followed the course described above. Vomiting was again an early sign, and continuous hiccup, which rapidly

produced extreme exhaustion, was noted in more than one case. The mental state varied from apathy and stupor to anxiety and delirium, which was notably resistant to sedatives. Pyrexia and augmented pulse-rate were constant, but, except when the injuries were very extensive, circulatory failure was less prominent than in children. We have, however, observed several adults with very extensive burns—up to 75 per cent of the body surface—in whom the disturbances from an early stage were predominantly those of circulatory failure. In 2 secondary shock was very severe and the response to treatment was incomplete, so that the blood-pressure did not regain a normal level, though life was prolonged till between 55 and 90 hours—that is, well into the stage of acute toxæmia. No doubt the stages of secondary shock and acute toxæmia overlapped and both conditions contributed to the fatal issue. Naturally, when the circulatory failure was profound between 6 and 24 hours, it was often impossible to judge whether it were due to secondary shock or early acute toxæmia. Another variation of acute toxæmia in adults was fairly frequent. A state of low-grade intoxication, marked by pyrexia and tachycardia, but without serious circulatory failure, was continued beyond the first week till bacterial infection supervened and proved an insupportable additional burden. In 2 such cases jaundice appeared at the 4th or 5th day. Investigation in adults gave results in all respects similar to those in children.

Incidence of Acute Toxæmia.—Several points which were noted regarding the incidence of acute toxæmia are worthy of record. It was the sole or main cause of death in 13 cases of the series and occurred in severe form in 4 others. It was more common and more severe in infants and young children than in adults. The relation between the extent of skin surface involved and the severity of acute toxæmia was much less constant than in the case of secondary shock. We have seen fulminating toxæmia in a small burn and very mild toxæmia in a burn of 60 per cent of the body surface. On the other hand, there was evidence of an association of toxæmia with a certain depth of injury, such as that produced by steam, boiling water, or a momentary exposure to flame. Cases with more superficial or with deeper burns frequently escaped toxæmia. Generally, in burns of moderate extent, the incidence of toxæmia was so irregular as to render impossible at the commencement of the course any prediction of its occurrence. If, however, the specific toxic manifestations had not appeared by 90 hours after injury, the chances of their development were remote.

4. SEPTIC TOXÆMIA

In regard to the stage of septic toxæmia, we shall deal separately with superficial and deep burns, since the incidence of infection was very intimately related to the depth of injury. The burns were classified as superficial when they involved only the skin, and as deep when they destroyed the whole thickness of skin and penetrated to deeper tissues. Usually scalds by hot liquid or steam were superficial, although we have observed destruction of the whole thickness of the skin over a large extent of surface by boiling water. After a burn by fire, a considerable proportion of the total area affected had often been deeply injured; nevertheless mining explosion burns were largely superficial.

Superficial Burns.—When incisions were made through the coagulum during the first week it was found that the coagulated layer was firmly incorporated with the œdematous underlying tissue; rarely did a collection of fluid intervene,

and, as already mentioned, there was seldom evidence of bacterial growth up to 90 hours. Subsequently various organisms such as streptococci and staphylococci appeared in cultures taken from certain portions of the burned areas, although both local and general signs of inflammation were often absent. If blisters formed in untanned areas, or if the coagulum became fissured or separated at the edges, then organisms, usually hæmolytic streptococci, could be grown from blister fluid or exposed raw surface; such conditions certainly provided an opportunity for bacterial growth. Infection was restricted to such points, and was denoted by reddening of the surrounding intact skin and exudation of fluid from the edge of the coagulum; the systemic disturbance, if any, was mild. Gross or dangerous infection of superficial burns was rare.

Deep Burns.—In the initial stages, and even during local treatment, it was often difficult to determine if the injury had destroyed the whole depth of skin. Sometimes deeply burned areas were indicated by the characters of the epidermis, which was scorched dark brown, toughened, firmly adherent, and therefore was unaffected by the application of tannic acid. If in deep areas the epidermis could be stripped off, a pale avascular tissue was exposed of which the surface layer was readily susceptible to the coagulating action of tannic acid, and the appearance subsequently was similar to that of superficial burns. Sometimes the main or only indication that the injury had penetrated deeply was the continued firm adherence of the coagulum for two or three weeks or longer. Separation of dead tissue, incorporated with the coagulated surface layer, was slow and was effected by the growth of granulation tissue, between which and the necrotic cells intervened a layer of yellow, inspissated serum containing leucocytes and cell debris. Bacterial invasion was heralded by local and general signs. The local signs were usually, though not invariably, obvious; the site of invasion was as a rule at the edge of the burn and, not infrequently, the inflammatory process was more extensive in the adjacent unburned tissues than in the burned area. Confirmation of this fact was obtained when attempts were made to remove the coagulum; a short distance from the inflamed area the coagulum was found everywhere firmly attached to healthy granulation tissue. Exceptionally a spreading cellulitic process invaded the tissues subjacent to the coagulum, by which the local inflammatory signs were obscured. The general systemic disturbance proved the more reliable index of bacterial activity. Even when the coagulum had become sodden and loose in parts, and large sloughs of necrotic tissue were being thrown off, revealing a thick purulent exudate of unpleasant aspect and odour, the general condition frequently remained satisfactory; in spite of some degree of pyrexia, the circulation was efficient and strength was unimpaired. Serious bacterial infection was usually caused by the hæmolytic streptococcus, and was denoted by a swinging temperature, rapid pulse, and, in the worst cases, signs of pyæmia. The organism was then found in cultures from burned area and blood.

In our series local signs of bacterial infection of deep burns were rarely present before the 5th day, and usually were not obvious till after the 7th day. Even in fatal septicæmia and pyæmia, hæmolytic streptococci did not appear in the blood before the 9th day. As regards incidence, if we assess bacterial infection by the results of cultures from the exudate beneath the coagulum and dead tissue, then infection was always present in deep burns during some part of the course. To judge by the general disturbance produced, the degree of infection in the majority

of cases was mild; in some, however, it was severe or very severe and fatal. Sepsis was the predominant cause of death in 10 cases of the series.

It was by no means always easy to determine the role of bacterial infection in systemic disturbances during the second week. Great difficulty was encountered in adults in whom a low-grade acute toxæmia had continued from an earlier period with a steady deterioration of strength culminating in prostration, stupor, or delirium, and sometimes incontinence of urine and fæces. The local signs of inflammation were often no more, or even less, obvious than in other cases without serious upset. Even at the post-mortem examination, in the absence of cellulitis or pyæmia it was impossible to judge whether death had been caused mainly by bacterial intoxication or by the effects of preceding or coincident acute toxæmia. As we shall show, diffuse destruction of liver cells was a characteristic lesion of acute toxæmia, and in all probability liver insufficiency was the main or an important contributory cause of death. No doubt even the mildest form of bacterial activity would in such instances have sufficed to turn the scales adversely.

As a point of practical interest, it may be mentioned that in 2 cases hæmolytic streptococcal infection, ultimately fatal, began in a granulating surface many weeks after the coagulum had been removed.

As in the case of acute toxæmia, it may be said that recent experience suggests that our conception of the course may shortly require modification. Since hæmolytic streptococcal infections can now be in large part prevented or successfully treated in deep burns, the cause of the systemic disturbances at the time when sloughs are separating may demand reconsideration. Till now we have attributed them to absorption of products of bacterial activity.

5. HEALING

This stage, which follows that of infection, has not been specially studied. Its importance will in the future be enhanced as therapeutic improvements serve to diminish the mortality in the earlier stages. Many surgical problems of great interest await attention.

COMPLICATIONS

It is convenient to consider separately the common complications of burning injuries.

Lesions of the Respiratory Tract.—The association of such lesions with burns of the chest has long been recognized, and it has been implied that they were in some way produced by the action of heat on the skin and tissues of the chest wall; the same association of inflammatory lesions of viscera with burns of overlying tissues has been claimed for other regions, such as the abdomen and head. In our experience the association held for burns of the chest, but not for those of the abdomen, and only in very exceptional circumstances for burns of the head, when the lesions were produced apparently by the direct action of heat on the brain itself. For instance, we have seen very severe, though temporary, cerebral disturbance in an infant following prolonged application of flame to the head; subsequently a large portion of the vault of the skull was extruded as a sequestrum.

Consideration of the facts in our cases of thoracic visceral lesions suggested an explanation of the association which seemed more simple and probable than the above. They were common after burns by fire which involved the anterior

aspect of the upper part of the trunk and the face, and particularly after accidents in which clothing had caught fire and some minutes had elapsed before the flames were extinguished. The lesions were common also after mining explosion burns, when the victims had been momentarily enveloped in a wall of flame and had subsequently breathed air at a very high temperature. On the other hand, if the burns involved only the posterior aspect of the thorax, respiratory tract lesions were unusual, and the same was true for scalds of any part of the thorax unless hot fluid or steam had been inhaled—by no means a rare accident in infants. Lesions of the respiratory tract, we consider, were caused by dirct injury to its lining membranes by inhalation of flame, hot air, or hot fluid. Admittedly the mucous membrane of nose and mouth usually escaped severe damage. Often the mucous membrane of pharynx, larynx, and trachea was also little affected, though we have observed in all these parts extensive sloughing lesions. The common respiratory lesions were, however, bronchitis, bronchopneumonia, and pulmonary œdema. They were particularly dangerous complications and an important contributory cause of death during the first and second weeks. Pulmonary œdema was a common lesion in cases of fatal acute toxæmia, and apparently was prone to develop when large quantities of fluid were administered intravenously, and particularly by the continuous-drip method.

Duodenal Ulcer.—Ulcers in the duodenum were found post mortem in 4 cases, one of which is quoted in detail among the illustrative cases. In only one instance was there any indication of the presence of ulceration during life, in this case hæmatemesis on the day before death. The victims were females, and the ages were 1 year 5 months, 7 years, 16 years, and 23 years. In all, the injuries were very extensive, and the time of death was between the 10th and the 12th day. In other respects, however, differences were present. In 3 the burns were deep, in 1 mainly superficial; 2 had hæmolytic streptococcal septicæmia and pyæmia, in 2 sepsis was inconspicuous. The liver cells were extremely and diffusely necrotic in 1, severely damaged in 2, but only slightly affected in the last. In no case was there histological evidence of special injury to the suprarenal gland or to the hypothalamus. In other cases of extensive burns, though not in any in which duodenal ulcers were subsequently found, we examined gastric secretion by test-meal at intervals during the first two weeks; total acidity was always reduced, and little or no free hydrochloric acid was found.

As regards causation, it would be useless at present to speculate. The ulcers were recent and were evidently a result of the burn. Their situation, on the posterior wall of the first part of duodenum, excluded the possibility that the direct action of heat on the viscus was responsible. Evidently bacterial infection was not essential for the production of the lesion, and it would seem that hypochlorhydria, and not hyperchlorhydria, was the usual sequel of extensive burns. In spite of negative histopathological evidence we cannot ignore the possibility that damage to the suprarenal gland or to the hypothalamus may have been responsible; damage to either may be followed by peptic ulceration.

Jaundice.—With increasing experience we have come to regard jaundice as one of the signs of acute toxæmia rather than a complication. It was noted in 12 cases of the series, and would probably have been detected more frequently in the earlier part of the investigation had its import been more fully realized. Jaundice was usually of the 'toxic' type; skin pigmentation was difficult to detect,

only traces of bile were found in the urine, the Van den Bergh reaction was biphasic, and the icteric index about 20 to 30. In several the jaundice was more definite and the urine contained much bile. In two cases 'latent' jaundice was present. Jaundice was found during fulminating toxæmia even as early as 48 hours after injury, but the most pronounced jaundice occurred in the slowly progressive low-grade toxæmia of adults, appearing usually about the 4th day. It was certainly not related to any special therapeutic measure, since it appeared under many modifications of treatment and was found in one late admission in which the burns had received no treatment beyond application of oil. Nor was it dependent on sepsis, since in most instances the presence of sepsis in the burned area was excluded. Jaundice indicated the occurrence of degenerative and necrotic changes in the liver.

Other Complications.—During the past ten years we have noted a steady diminution in the incidence of complications such as scarlatina and gastro-enteritis, which at one time frequently supervened during the course of burns. No doubt scarlatina was an indication of streptococcal infection of the burned area, which can now be more effectively controlled. In our series these complications were almost entirely restricted to untreated or badly-treated cases, for which admission to hospital was sought solely on account of the complication. We have already mentioned the common cerebral manifestations of acute toxæmia, such as delirium and epileptiform convulsions. One unique sequel of a small burn, in a child previously healthy, was extensive intravascular clotting, which involved not only the intracranial venous sinuses and Rolandic veins, but also the arteries of the lower extremities from the level of the lower part of the abdominal aorta downwards.

RAPIDLY FATAL BURNS OF MODERATE OR SMALL EXTENT

Most surgeons of experience can recall one or more instances of unexpectedly rapid death after a small burn, and this feature alone has impressed many with the potential dangers of such injuries.

Two examples have been encountered in our series. One (*Case* 9) is described among the illustrative cases. The other, a male child aged 3 years 6 months, died 24 hours after an injury involving only 5 per cent of the total skin surface; no treatment of any kind had been carried out. Since both died immediately after admission to hospital, clinical investigation was impossible. There was in both a striking pathological change, namely, infiltration of the liver by eosinophil leucocytes, which will be described later. The association of such a change with a rapidly fatal outcome suggested the possibility of anaphylaxis, and it is of interest that the second child had sustained a burn one year previously. The possibility is a mere conjecture, and is mentioned here in order that others may be led to inquiry on the point.

PATHOLOGY

The pathological investigation was carried out on 33 fatal cases, in 20 of which a study of the clinical course had been made.

Death within 24 hours.—Six cases of death within 24 hours of injury were available for examination. Three of these were under our own care and have already been mentioned, 1 a very rapid death after burns of moderate extent, and 2 cases of severe initial shock. We have received sufficient clinical data for a rough

analysis of the others; 2 had sustained very extensive injuries and died from initial or secondary shock within a few hours; 1 is described later (*Case* 5).

The pathological feature of note in 2 cases of rapid death (*see Table I*) was infiltration of the liver and other organs with eosinophil leucocytes, which was evidence of some unusual process. In the remaining cases, those of fatal initial or secondary shock, the changes were neither conspicuous nor specially distinctive. Pallor was the outstanding feature in most organs, though some, such as the brain, lung, and spleen, were sometimes moderately congested. Pulmonary œdema was a common occurrence and was most pronounced when intravenous infusions had been given. Examination of the injured surface revealed some surprising features. In superficial lesions the surviving dermis frequently showed little or no cellular infiltration, hyperæmia, or even œdema. Œdema, if present, was more obvious in the subcutaneous tissues than in the dermis. On occasion injury by scalding produced lesions of unexpected depth; we have observed coagulative necrosis of the whole thickness of the skin by a scald. At 21 hours after injury there appeared the earliest signs of the characteristic change of liver degeneration.

Death between 24 *and* 100 *hours.*—Our material includes 14 cases which terminated fatally during this period. Eleven patients who were personally observed died solely or mainly from acute toxæmia; in 2 of the remainder secondary shock probably accounted for death. In general, therefore, this period may be regarded as that during which severe acute toxæmia proved fatal.

In only one case was there evidence of microbial infection of the burned area; the pathological changes were therefore characteristic of acute toxæmia of burns. The most striking features were found in the liver; apart from this, no organ showed any constant or remarkable change. Some degree of fatty degeneration was common in the kidney and occasional in the myocardium. Changes in the *suprarenal glands* were carefully looked for; they were, however, very inconstant, and consisted of varying degrees of congestion, which when pronounced was associated with slight hæmorrhage, and of nuclear degeneration of the cortical cells. On occasion multiple erosions of the gastric mucosa were found, to which vomiting of altered blood during toxæmia was to be attributed. The other organs were affected in much the same way as in individuals dying before 24 hours. In most instances practically the whole of the burned area was free from any sign of bacterial growth.

Death after 100 *hours.*—Thirteen cases in which death occurred between 6 days and 6 weeks were examined. Five were examples of prolonged acute toxæmia without definite signs of severe bacterial infection; the presence of sepsis was doubtful or at most the degree of infection was very mild. In the remaining 8 cases sepsis was severe or very severe.

In prolonged acute toxæmia the changes were very similar to those described for rapidly fatal toxæmia: in 2 instances there were also duodenal ulcers. In cases of sepsis the changes were those of septicæmia or pyæmia. Congestion and hæmorrhage in the suprarenal gland, though inconstant, were more common in this group than in cases of earlier death.

Liver Necrosis and Degeneration.—This, the most striking and characteristic feature of the pathology of burns, has not, so far as we can find, received special attention in the past.

In its earliest form, at 21 hours after injury, it appeared as fatty degeneration of the epithelial cells surrounding the efferent veins in the central zone of the

hepatic lobules; the fatty degeneration was accompanied by nuclear damage, especially karyolysis, which was constant and was more severe in proportion to

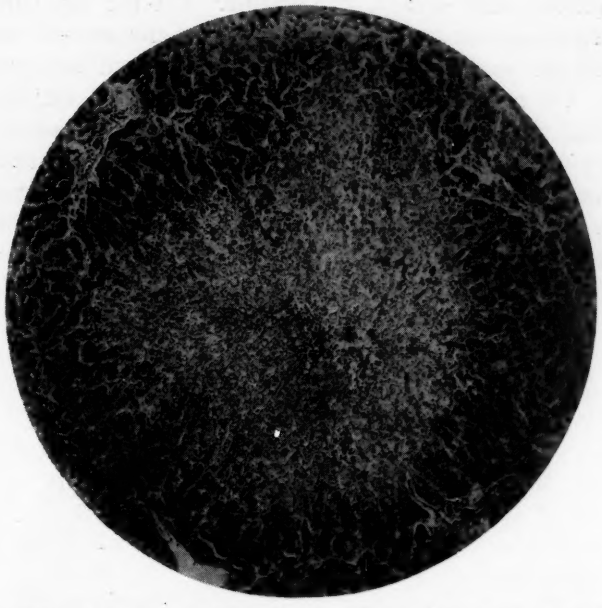


FIG. 606.—Microphotograph of liver showing degeneration and necrosis of cells of central zone of lobule. Death at 100 hours from acute toxæmia. (× 50.)

FIG. 607.—Microphotograph of liver showing extreme fatty degeneration of cells of central zone. Death at 68 hours from acute toxæmia. (× 160.)

cytoplasmic changes than in most other conditions, such as anæmia and chronic venous congestion, which produce degeneration of cells of the central zone. Evidently even at this early stage the cells had suffered serious injury.

At a more advanced stage, from 57 hours onwards, the characteristic severe form of damage was found. On naked-eye examination the liver was slightly enlarged, light yellow, soft, greasy, and friable. On the cut surface the lobular marking was conspicuous owing to greater pallor of the central zone; sometimes, however, hæmorrhage in the central zones produced an appearance resembling 'nutmeg' liver. On microscopic examination necrosis of varying degree of the cells of the central parts of the lobules was seen (*Figs.* 606–608). In the most extreme examples only a narrow strip of liver substance surrounding each portal tract showed surviving liver cells; the remainder were completely necrotic. Sections suitably stained showed much free fat in the dead cells, but the most completely necrotic cells of the central zones might contain less fat than those less severely damaged nearer the periphery of the lobules. The disorganization of

FIG. 608.—Microphotograph of liver showing extreme degeneration and necrosis. Death at 71 hours from acute toxæmia. (× 60.)

liver structure was such that with collapse of the walls of the sinusoids hæmorrhage occurred in the central zones, thus producing, as already mentioned, a condition simulating the 'nutmeg' liver of chronic venous congestion. It was clear, however, from consideration of the earlier changes, that the process was primarily a degeneration leading to necrosis of the parenchyma cells. In the extent and completeness of destruction of liver tissue, the microscopic picture was comparable with that of acute liver atrophy, but differed in the zonal distribution, which was constant and characteristic. It differed also from the majority of bacterial toxæmias both in affecting the central instead of the peripheral zone and in the more pronounced severity of the injury to the liver cells.

In *Table I* the relation of the liver lesion to the incidence and severity of acute toxæmia is shown. Cases not personally observed by us and cases admitted late with grossly infected burns have been marked as yielding insufficient data; in one

Table I.—THE RELATION OF ACUTE TOXÆMIA AND SEPSIS TO LIVER CHANGES IN 33 FATAL CASES

INITIALS	SEX	AGE IN YEARS	TIME OF DEATH	ACUTE TOXÆMIA	SEPSIS	LIVER	
						Degeneration	Eosinophil Cells
G. B.	M.	1 yr. 7 mth	5½ hr.	—	—	—	Numerous
M. P.	F.	2 yr. 10 mth.	7½ hr.	—	—	—	Numerous
T. W.	M.	57 yr.	9 hr.	—	—	—	—
J. F.	F.	50 yr.	11 hr.	—	—	—	—
A. McL.	F.	2 yr. 10 mth.	21 hr.	—	—	Slight	—
G. M.	M.	7 yr.	23 hr.	?	—	Slight	—
R. B.	M.	3 yr. 6 mth.	24 hr.	I. D.*	—	Slight	Present
C. M.	F.	21 yr.	24 hr.	I. D.	—	Slight	—
G. H.	M.	1 yr. 7 mth.	36 hr.	I. D.	—	—	Present
E. T.	F.	8 yr.	57 hr.	Severe	—	Severe	Present
N. T.	F.	20 yr.	60 hr.	Severe	—	Slight	—
A. R.	F.	10 yr.	63 hr.	Severe	—	Very severe	—
F. G.	F.	5 yr. 6 mth.	68 hr.	Severe	—	Severe	—
R. O'D.	M.	9 mth.	68 hr.	Severe	—	Severe	—
J. R.	F.	1 yr. 10 mth.	68 hr.	Severe	—	Severe	—
R. G.	M.	9 mth.	71 hr.	Severe	Present	Very severe	—
J. B.	M.	8 mth.	78 hr.	Severe	—	Severe	—
J. M.	F.	50 yr.	80 hr.	Severe	—	Slight	—
E. S.	F.	9 yr.	93 hr.	Severe	—	Fairly severe	—
M. A.	M.	30 yr.	100 hr.	Severe	—	Very severe	—
T. S.	M.	6 yr.	6 d.	I. D.	Severe	—	Present
A. O.	M.	1 yr.	7 d.	I. D.	Severe	—	—
G. McL.	F.	2 yr. 3 mth.	8 d.	Severe	Present	Severe	—
P. A.	M.	34 yr.	8 d.	Prolonged	Severe	Severe	—
G. G.	F.	1 yr. 11 mth.	9 d.	I. D.	Severe	Slight	Present
A. J.	F.	7 yr.	10 d.	Severe	—	Severe	—
C. S.	F.	1 yr. 5 mth.	11 d.	Mild	Very severe	—	—
B. R.	F.	23 yr.	11 d.	Prolonged	Very mild	Very severe	—
J. W.	F.	16 yr.	12 d.	Severe	Severe	Severe	—
M. C.	F.	3 yr. 6 mth.	14 d. (Accidental death)	Fairly severe	—	Recovering	—
C. McG.	M.	1 yr. 10 mth.	15 d.	I. D.	Very severe	—	—
G. W.	M.	2 yr. 5 mth.	20 d.	Moderate	—	—	—
B. M.	F.	12 yr.	6 w.	Mild	Severe	—	—

* I.D. = Insufficient data. Cases not specially investigated.

other case the diagnosis of toxæmia was doubtful. Omitting these from consideration, it will be seen that the relationship of the liver lesion to acute toxæmia was in every respect very close. Of 16 cases of severe or prolonged acute toxæmia, the liver suffered intense damage in 14. In cases of toxæmia without severe liver damage, treatment had included continuous intravenous dextrose saline from an early stage, a point to which we shall refer in discussing treatment.

Evidently, also, bacterial infection was not responsible for the liver lesions. In the majority of cases with liver damage there was no definite evidence of infection. Moreover, the typical lesion was not produced by sepsis even in its worst form; in sepsis the changes in the liver were those usual after grave bacterial infections,

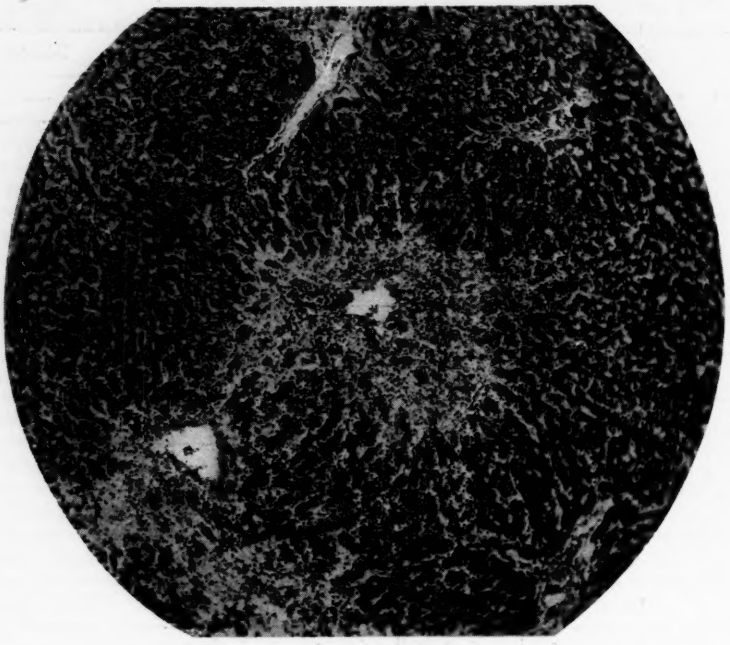


FIG. 609.—Microphotograph of liver showing mild injury with recovery of surviving cells. Accidental death on 14th day. (× 65.)

but were incomparably less serious than the changes we have described as characteristic for burns. Lastly, when liver lesion and sepsis coexisted, the severity of one bore no relation to the severity of the other.

That some degree of liver damage was sustained in a number of patients who survived was indicated by the occurrence of jaundice. Pathological confirmation was afforded by a case of accidental death on the 14th day after extensive and deep burns. The main features of the course had been profound secondary shock, moderately severe acute toxæmia, and jaundice between the 3rd and 9th days; there was no obvious bacterial infection and the outlook had been favourable. In the liver (*Fig.* 609) the cells in the centre of each lobule had disappeared more or less completely, leaving only a reticulum with a few remnants of necrotic cells, and the surrounding liver cells were healthy. The obvious interpretation was that an injurious agent which had destroyed many cells had ceased to be active at the time of death and that such cells as had survived had returned to normal.

In summarizing we may affirm that after death from burns a lesion of the liver cells was found in many cases which was characteristic of this form of injury. Its relation to acute toxæmia was so remarkably close as to leave little doubt that liver lesion and acute toxæmia were produced by the same mechanism. The responsible agency was certainly not bacterial infection, and, in our view, the liver lesion furnished the strongest indication of a non-bacterial toxin circulating during the first few days after a burn.

Eosinophil Cell Infiltration.—This feature has not to our knowledge been mentioned previously in pathological descriptions. We found it in 7 fatal cases (*Table I*). The most striking examples were in 2 cases of early death ($5\frac{1}{2}$ and $7\frac{1}{2}$ hours after injury). In these the accumulations of eosinophil leucocytes were large enough to be visible to the naked eye as bright yellow spots of pin-head size under the capsule of the liver and on the cut surface; microscopically they presented a remarkable appearance (*Fig.* 610) of dense masses of leucocytes crowding the

FIG. 610.—Microphotograph of liver showing infiltration by eosinophil leucocytes. Death at $7\frac{1}{2}$ hours. (× 50.)

tissue, especially the fibrous tissue of certain portal tracts, and associated in one instance with small foci of necrosis. In the remainder the infiltration was much less massive and was found only on microscopic examination; there was also some excess of eosinophils in other situations, particularly the spleen and lymph-glands. The infiltration was found as late as 9 days after injury, and evidently was not always associated with a rapidly fatal issue. As mentioned previously, we have found no eosinophilia in the blood in our series. At present we are unable to express an opinion as to the significance of the observation.

DESCRIPTION OF ILLUSTRATIVE CASES

The following cases have been selected to illustrate important clinical and pathological features on which our main conclusions in the foregoing sections have been based.

FIG. 611.—*Case* 1. Photograph showing extent of lesion.

Case 1.—**Extensive scalds. No initial or secondary shock; mild acute toxæmia; very mild sepsis; recovery.**

E. H., male, aged 11 years, admitted 2 hours after injury. Lesions healed by 30th day. Extent shown in *Fig.* 611. Clinical course during first 12 days illustrated by *Chart* 1.

POINTS.—(1) High leucocyte count on admission. (2) During mild acute toxæmia circulatory efficiency was unimpaired, there was no change in blood concentration or sedimentation rate, and no evidence of bacterial infection; changes in blood chemistry were insignificant. (3) Jaundice from 4th to 9th day following toxæmia. (4) Very mild sepsis from 8th to 12th day.

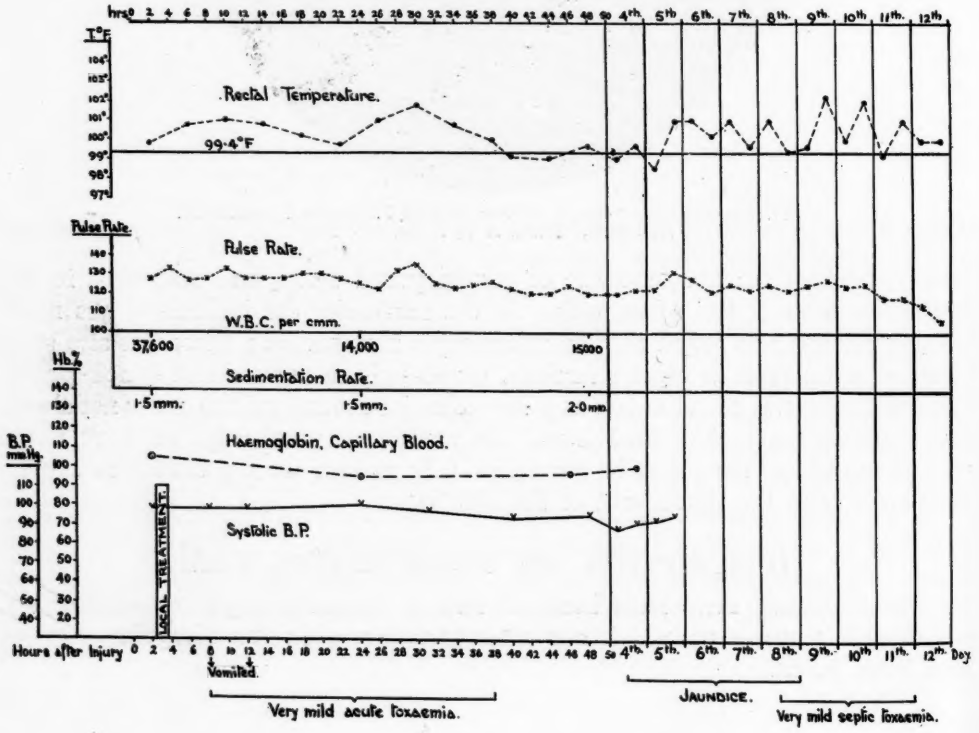
CASE 1.
BLOOD CHEMISTRY.

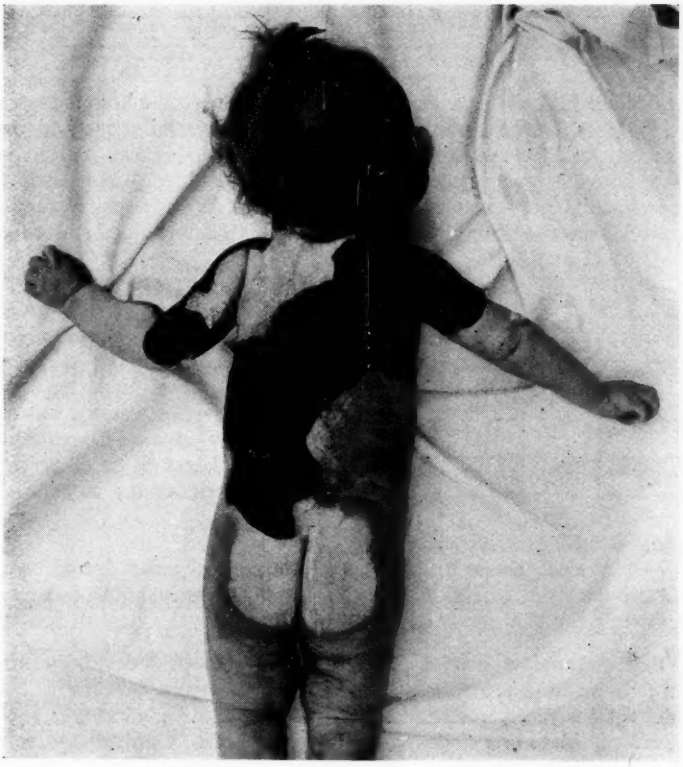
Hours	CO_2 vols. %	Cl mgm. %	Plasma Proteins gms. % Alb.	Glob.	Serum Ca mgm. %	N.P.N. mgm. %
2½	44	520	4·55	1·82	10·6	36
26	46	448	4·85	2·07	11·8	45

BACTERIOLOGY. All cultures from burned area up to 70 hours were sterile.

Comment.—A fairly typical example of mild acute toxæmia and mild sepsis.

Chart 1. Case 1.



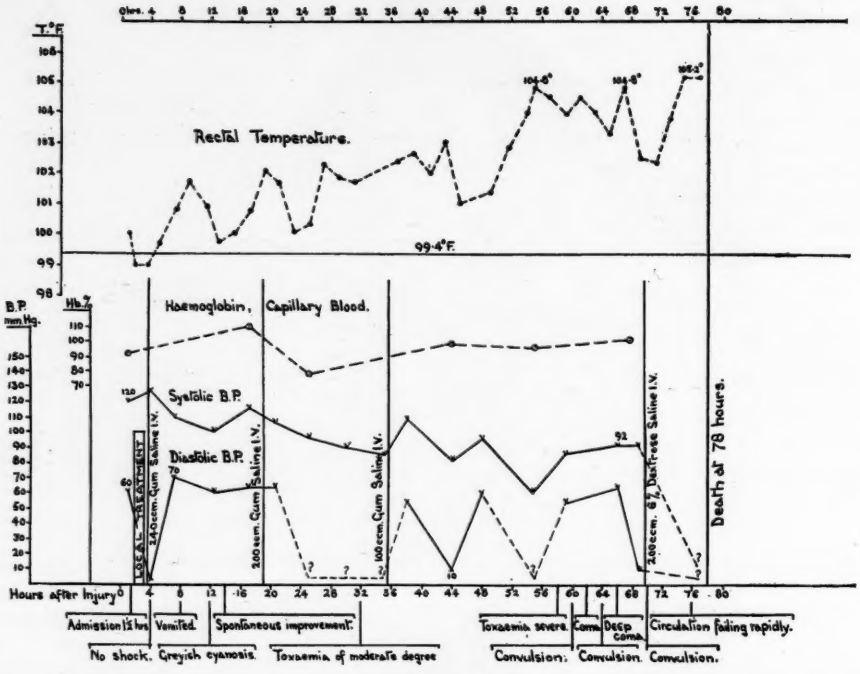


FIGS. 612, 613.—*Case* 2. Photographs showing extent of scalds.

Case 2.—**Moderately extensive scalds. No initial or secondary shock; early fulminating acute toxæmia; death at 78 hours.**

J. B., male, aged 8 months, admitted at 1½ hours after injury. Extent shown in *Figs.* 612, 613. Clinical course illustrated by *Chart* 2.

Chart 2. Case 2.



POINTS: (1) No initial or secondary shock. (2) Onset of acute toxæmia at 8 hours; early progressive pyrexia; severe cerebral disturbance with restlessness, apathy, stupor, and terminal coma punctuated by repeated convulsions. (3) No serious fall in systolic blood-pressure till a few hours before death; erratic diastolic level. (4) No conspicuous increase in blood concentration; very evanescent improvement after each intravenous infusion. (5) No evidence of bacterial infection. (6) Moderate fall in blood-chlorides at 9 hours before death.

Case 2.

BACTERIOLOGY: CULTURES.

Hours	Burned Area.		Blood.	
	Aerobic	Anaerobic	Aerobic	Anaerobic
54.	Sterile.	Sterile.	Sterile.	Sterile.
78.	Sterile.	Sterile.	Sterile.	Sterile.
Post-mortem	Sterile.	Sterile.	Contaminants.	

BLOOD CHEMISTRY.

Hours.	Cl mgm. %	Plasma Proteins gm. %	
		Alb.	Glob.
2½	455.	3·2	3·7
17½	455	3·3	3·6
69	395.	—	—

POST-MORTEM EXAMINATION.—

Liver.—Large, pale, 'nutmeg' appearance. Severe degeneration and necrosis of cells in central zones of lobules; hæmorrhagic congestion in parts.

Lungs.—Congestion, some œdema.

Injured Area.—Coagulative necrosis of whole thickness of skin, no cellular infiltration, no hyperæmia; œdema of subcutaneous tissues.

Other Organs.—Nothing of importance.

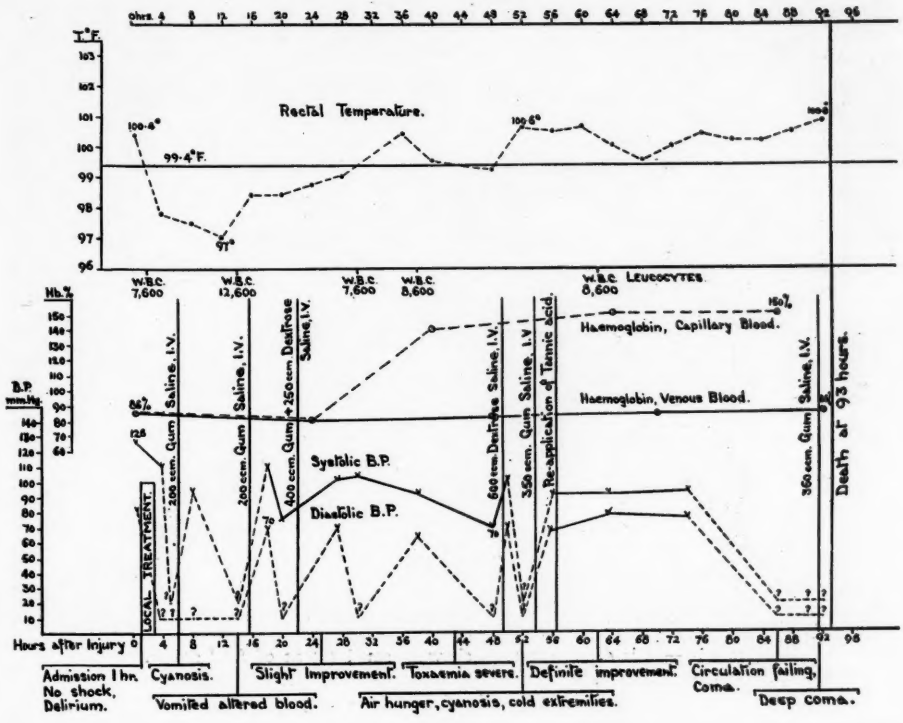
Comment.—A typical example of early fulminating acute toxæmia. Anhydræmia, hypochloræmia, and bacterial infection were excluded as aetiological factors. Some other agency was responsible for the overwhelming illness and for the destruction of liver cells.

Case 3.—**Very extensive burns. No initial shock; secondary shock; severe acute toxæmia; bronchopneumonia; death at 93 hours.**

E. S., female, aged 9 years, admitted 1 hour after injury. Extent was about 65 per cent of total body surface, including face and front of chest. Clinical course illustrated by *Chart* 3.

POINTS: (1) No initial shock even after very severe and extensive burns. (2) Secondary shock after local treatment; tendency for blood-pressure to fall remained for 24 hours. Probably overlapping of secondary shock and onset of acute toxæmia. (3) During acute

Chart 3. Case 3.



toxæmia there were periods of collapse with sudden falls of blood-pressure. (4) The capillary blood became concentrated, evidently as a result of local circulatory stasis; the venous blood was unaltered. (5) No evidence of bacterial infection; no leucocytosis. (6) No evidence of acidosis and no fall of blood-chlorides.

POST-MORTEM EXAMINATION.—

Trachea and Bronchi.—Acutely inflamed.

Lungs.—Extensive acute bronchopneumonia. These respiratory tract lesions were associated with burns of face and front of chest.

Liver.—Severe degenerative changes in central zones of all lobules; many necrotic cells.

Kidney.—Degeneration of epithelium of tubules.

Case 3

BACTERIOLOGY: CULTURES.

Hours	Burned Area. Aerobic	Burned Area. Anaerobic	Blood.
19	Sterile	Sterile.	——
40	Sterile	Sterile.	——
80	Sterile	Sparse growth of streptococci.	——
93	——	——	Sterile.
Post-mortem	——	——	Streptococci, Coliform bacilli.

BLOOD CHEMISTRY.

Hours	CO_2 vols. %	Cl mgm. %	Plasma Protein gm. % Alb.	Plasma Protein gm. % Glob.	Serum Ca. mgm. %
1½	—	540	4·3	3·0	12·0
47	41·3	—	—	—	—
70	41·8	450	—	—	—
93	43·5	530	2·0	3·5	11·7

Burned Area.—A narrow band of dense leucocytic infiltration deep to the coagulum. In deepest part of cutis vera and subcutaneous tissues no cellular infiltration or œdema and little hyperæmia.

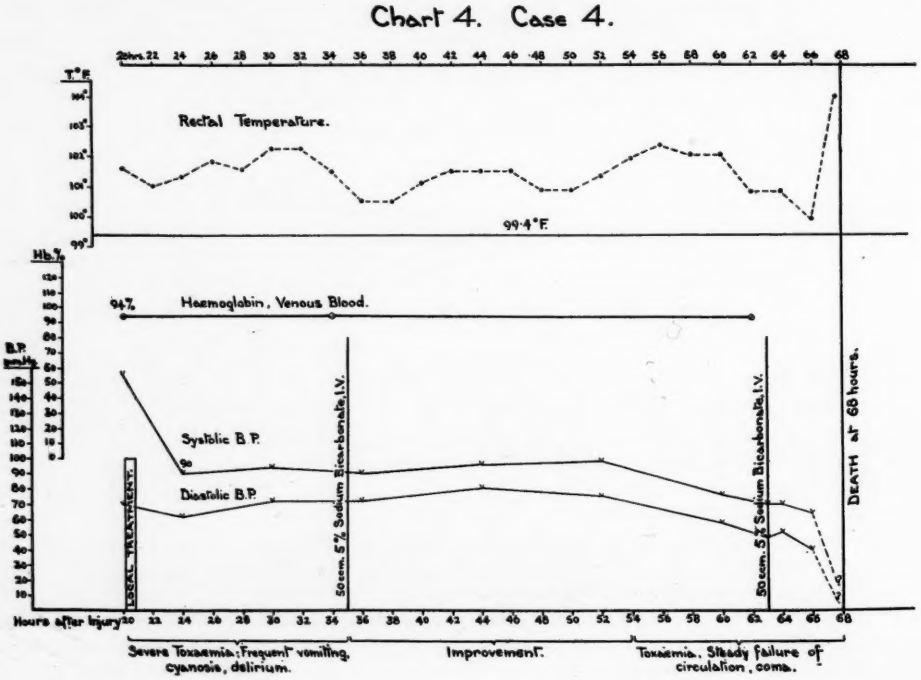
Other Organs.—Nothing of importance.

Comment.—Another example of early severe acute toxæmia which was not due to anhydræmia, bacterial infection, or changes in blood chemistry. Respiratory tract lesions were a contributory cause of death.

Case 4.—**Scalds of small extent. Severe acute toxæmia on admission; death at 68 hours.**

J. R., female, aged 1 year 10 months, admitted at 20 hours after injury. Toxæmia had begun at 8 hours after injury. Extent shown in *Fig.* 614. Clinical course illustrated by *Chart* 4.

FIG. 614.—*Case* 4. Photograph showing small extent of fatal scalds.



POINTS: (1) Gravely ill on admission. Toxæmia not controlled by tannic acid treatment. Apparent temporary benefit from intravenous sodium bicarbonate. (2) Steady slow fall in blood-pressure during the last 16 hours. (3) No increased blood concentration, no evidence of bacterial infection, and, apart from some fall of blood-chlorides, no change in blood chemistry.

POST-MORTEM EXAMINATION.—

Liver.—Pale, yellow, greasy. Extreme fatty degeneration of all parts of the lobules, early necrosis of cells in central zones.

Kidney.—Widespread fatty degeneration of the cells of all tubules.

Heart.—Fatty degeneration of inner part of myocardium.

Lungs, Spleen, Suprarenals.—Congestion.

Other Organs.—Nothing of importance.

Case 4.

BACTERIOLOGY: CULTURES.

Hours	Burned Area.		Blood.	
	Aerobic	Anaerobic	Aerobic	Anaerobic
Post-mortem	Sterile	Sterile	Sterile	Sterile

BLOOD CHEMISTRY.

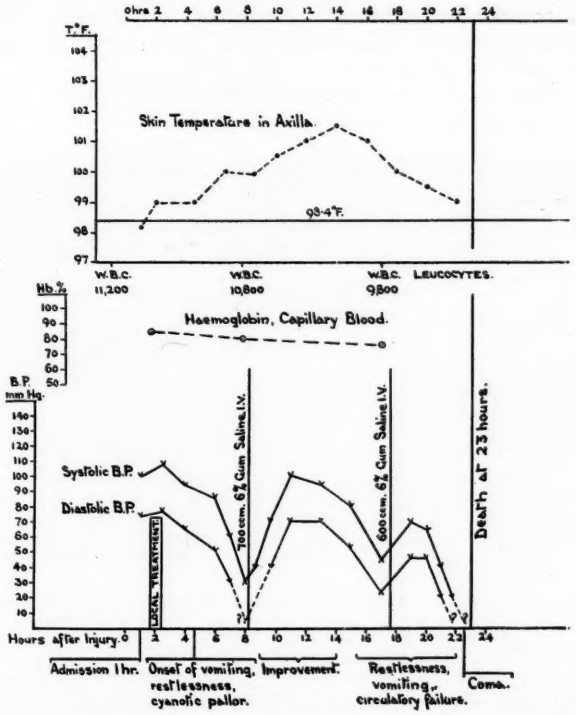
Hours	CO_2 vols. %	Cl mgm. %	Plasma Protein gm. % Alb.	Glob.	Serum Ca. mgm. %
34	49·4	457	—	—	10·6
62	50·5	369	4·56	2·08	9·55

Comment.—An example of early, severe, and fatal acute toxaemia following a small injury. Degenerative changes in the organs were widely distributed, the liver being most affected. Anhydræmia, bacterial infection, and significant changes in blood chemistry were absent. The failure to respond to tannic acid treatment was unusual in such a small injury.

Case 5.*—Scalds of moderate extent. Treatment by a weak tannic acid preparation; death at 23 hours from persistent secondary shock or fulminating acute toxæmia or both.

G. M., 7 years, admitted at 1 hour after scalds of both legs. Local treatment was the application of jelly containing 5 per cent tannic acid and a mercurial antiseptic. Clinical course illustrated by *Chart* 5.

Chart 5. Case 5.



* The investigation and treatment of this case were carried out by Dr. G. D. Rowley, to whom we are indebted or details and for permission to publish.

POINTS.—(1) No initial shock. (2) Persistent tendency to fall in blood-pressure after 4½ hours. The response to intravenous gum saline was only evanescent at 8 hours and failed altogether at 18 hours. (3) Pyrexia and other features of the clinical course were strongly reminiscent of acute toxæmia. (4) No change in blood concentration or chemistry; no bacterial infection.

Case 5.

BACTERIOLOGY: CULTURES.

Hours	Burned Area.		Blood.	
	Aerobic	Anaerobic	Aerobic	Anaerobic
1	Sterile.	Sterile.	Sterile.	Sterile.
8	—	—	Sterile.	Sterile.
17	—	—	Sterile.	Sterile.
20½	Sterile.	Sterile.	—	—
22½	—	—	Sterile.	Sterile.

BLOOD CHEMISTRY.

Hours	CO_2 vols. %	Cl mgm. %	Plasma Protein gm. %	
			Alb.	Glob.
1	58	680	4·2	1·9
8	63	640	4·0	1·7
17	61	600	4·2	1·7
22½	61	600	4·2	1·7

Urine: no abnormal constituents.
Differential blood film (14 hours): slight increase in eosinophils.

POST-MORTEM EXAMINATION.—Beyond slight degeneration of the cells of the central zones of the liver lobules, there was no change of note. There was nothing to suggest mercurial poisoning.

Comment.—This case is included here because the clinical course, though resembling that frequently described for fatal burns, has been outwith our experience in the present series. It is possible that the unusual course was the result of the use of a preparation with a slow coagulating action. The balance of evidence would appear to favour the view that death was due mainly to fulminating toxæmia. Anhydræmia, bacterial infection, and changes in blood chemistry certainly played no part.

Case 6.*—**Scalds of moderate extent. No initial or secondary shock; fulminating acute toxæmia which steadily regressed when extract of suprarenal cortex was administered; no sepsis; recovery.**

D. M., male, 3 years 2 months, admitted 2 hours after injury. Extent shown in *Fig.* 615. Clinical course up to 120 hours illustrated by *Chart* 6.

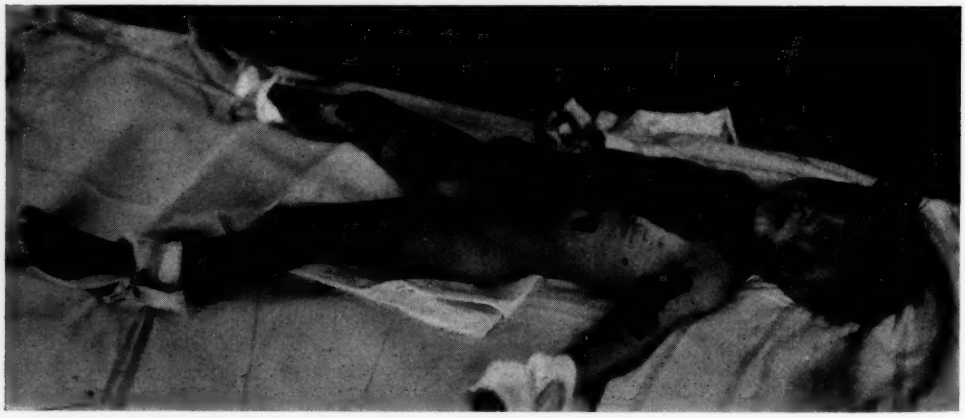


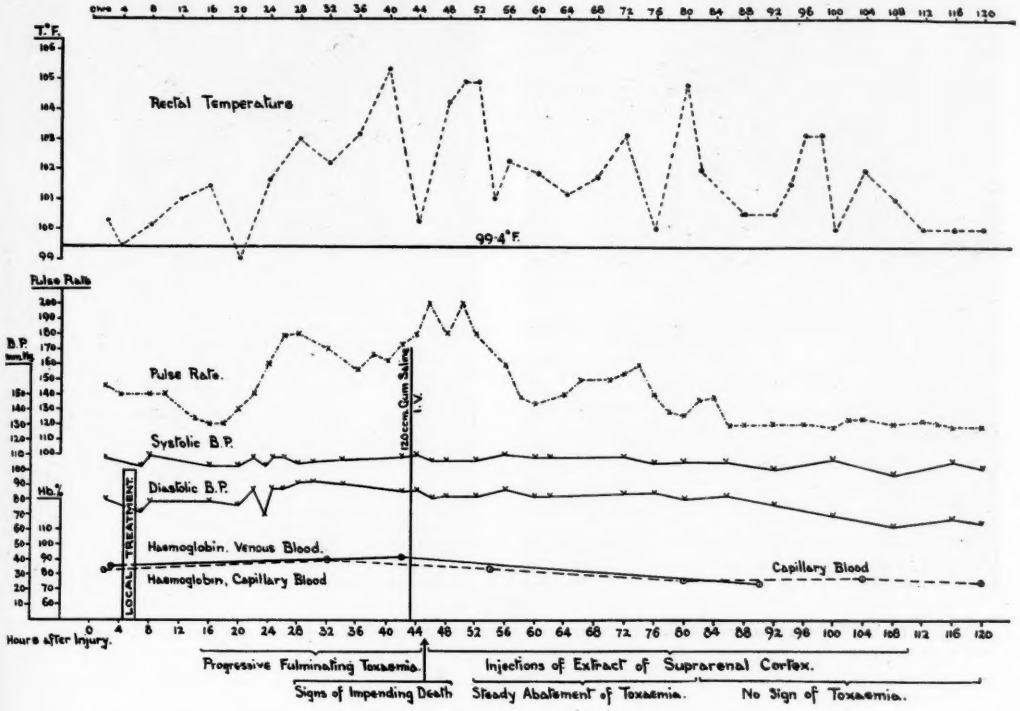
FIG. 615.—*Case* 6. Photograph showing moderate extent of lesions.

POINTS.—(1) No initial or secondary shock. (2) Acute toxæmia of fulminating type; diminished pulse pressure, increased heart-rate, pyrexia. By 45 hours pronounced circulatory failure and coma. (3) Toxic signs receded steadily when administration of extract of suprarenal

* This case has been mentioned briefly in a previous publication.[32]

cortex was begun. (4) During development and also regression of toxæmia there was no significant change in blood concentration or blood chemistry; no evidence of bacterial infection up to 90 hours. (5) Diminution of early leucocytosis irrespective of toxæmia.

Chart. 6. Case 6.



BACTERIOLOGY: CULTURES.

Hours	Burned Area. Aerobic.	Anaerobic.
3	Sterile.	Sterile.
24	Sterile.	Sterile.
40	Sterile.	Sterile.
67	Sterile.	Sterile.
90	From Tanned Area Sterile. / Sterile. From Isolated Blister Haemolytic streptococci	

Case 6.

BLOOD CHEMISTRY.

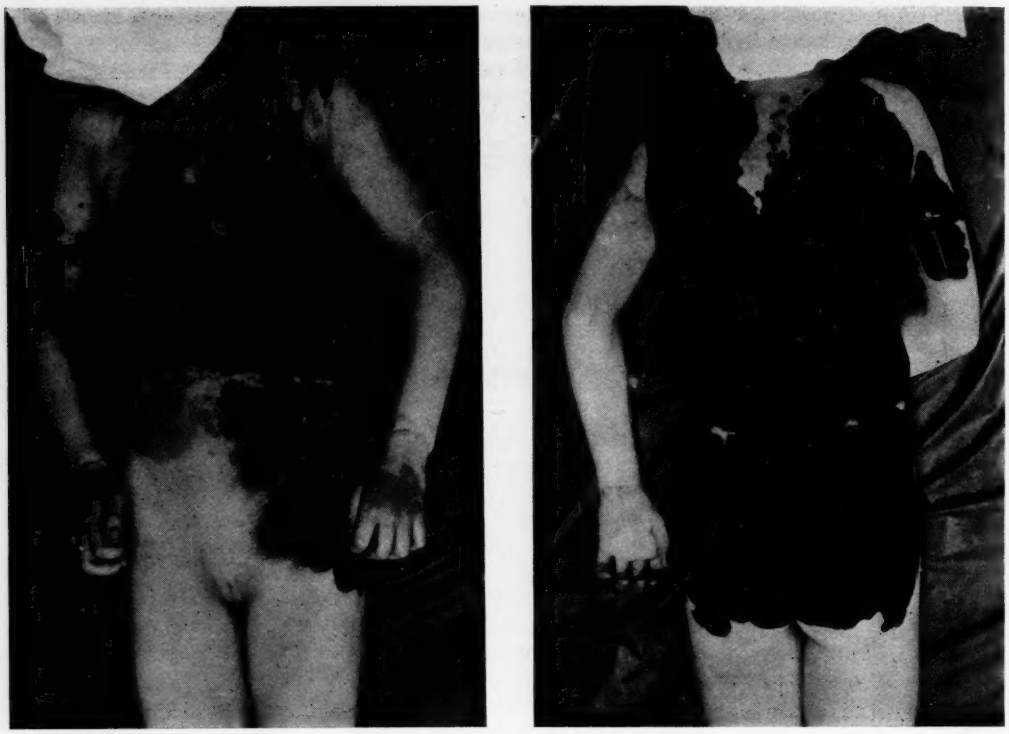
Hours	CO_2 vols. %	Cl mgm. %	Plasma Proteins gms. % Alb.	Glob.	Serum Ca. mgm. %	N.P.N. mgm. %	Cholesterol mgm. %
4	53	Plasma 640	—	—	12·3	—	—
42	53	Blood 558	4·05	2·15	10·3	30	160
90	47	Blood 495	4·30	1·92	12·0	36	—

Urine: Acetone between 24 and 80 hours. Otherwise no abnormal constituent.

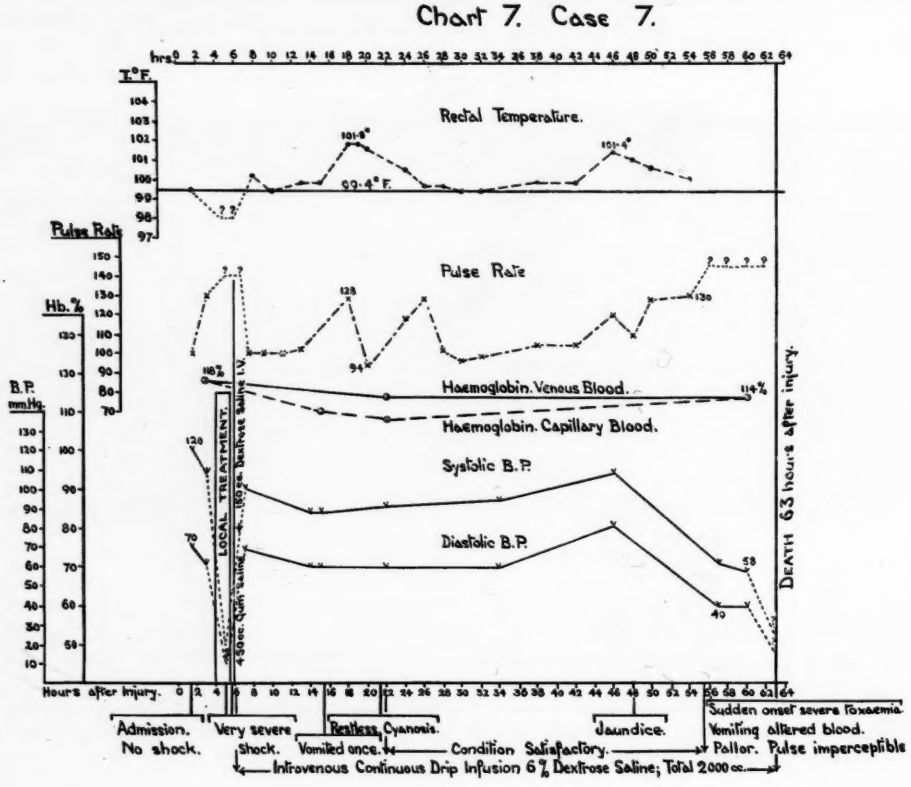
BLOOD SEDIMENTATION RATE AND LEUCOCYTES.

Hours	Sed. rate mm. per hour.	W.B.C. per cmm.	Neutrophils.	Large Lymphocytes.	Small Lymphocytes.	Monocytes.	Myelocytes.	Eosinophils.
4	4	20,000	75%	3·5%	20·5%	0	0·5%	0
32	2	14,600	65%	11·%	16·%	4	3·%	0
54	27	11,200	61%	4·5%	34·5%	0	0	0
80	34	9,000	—	—	—	—	—	—
104	27	6,800	—	—	—	—	—	—
128	27	8,000	—	—	—	—	—	—

Comment.—The action of extract of suprarenal cortex, which was exerted apparently mainly on the circulatory mechanism, was in this case dramatic. Apparently the liver and other important organs escaped irreparable damage.



FIGS. 616, 617.—*Case* 7. Photographs, taken post mortem, showing very extensive area of lesions.



Case 7.—**Very extensive scalds. No initial shock. Severe secondary shock. Late onset of rapidly fatal acute toxæmia.**

A. R., female, 10 years, admitted at 1½ hours after injury. Extent shown in *Figs.* 616, 617. Clinical course illustrated by *Chart* 7.

POINTS: (1) No initial shock even after very extensive scalds. (2) Very severe secondary shock developed during local treatment; rapid response to treatment. (3) Intravenous continuous-drip infusion of dextrose-saline throughout. (4) Sudden and unexpected onset of toxic signs at 56 hours; extract of suprarenal cortex then given intravenously was without benefit. Rapidly fatal toxæmia. (5) Early appearance, at 48 hours, of jaundice. (6) Serum sodium low before shock and again during toxæmia; chlorides and urea-nitrogen unaltered. (7) Blood concentration moderately raised throughout.

POST-MORTEM EXAMINATION.—

Liver.—Intense diffuse necrotic and degenerative changes.

Stomach.—Numerous erosions of mucosa—the cause of hæmatemesis.

Lungs.—Œdema pronounced.

Kidney.—Widespread degeneration of tubular epithelium.

Other Organs.—Nothing of importance.

Case 7.
BLOOD CHEMISTRY.

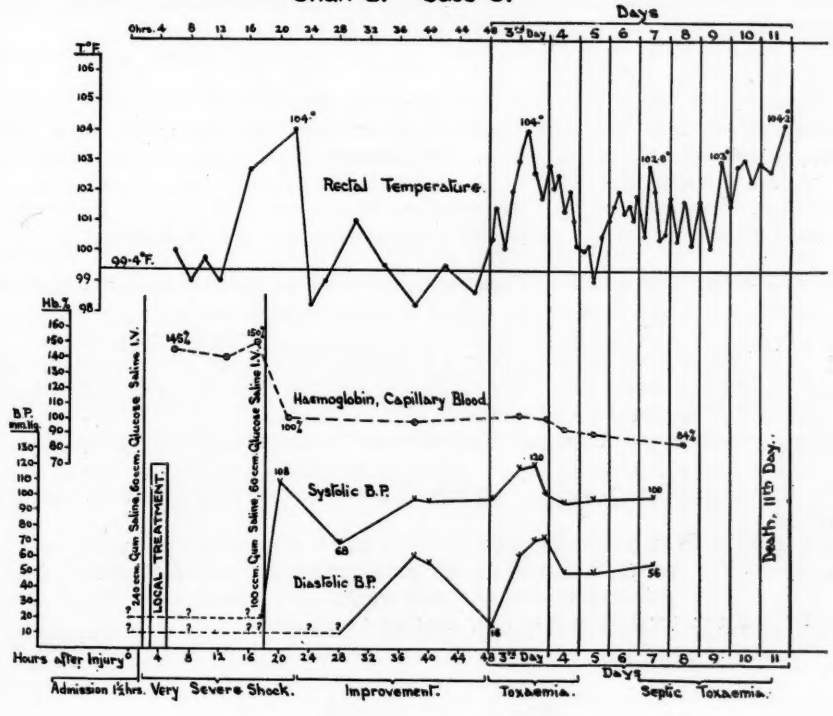
Hours	Hb. %	Serum Na mgm. %	Urea N. mgm. %	Chlorides mgm. %	Remarks.
3½	118%	279	12	484	Before shock.
23	114%	312	10	480	Condition satisfactory.
60	114%	251	14	496	Severe Toxaemia.

URINE.

Hours	Chlorides	Albumen	Casts	Blood	Sugar	Bile	Leucine + Tyrosine	Acetone
18	+	neg.	neg.	neg.	neg.	neg.	—	neg.
48	+	Trace	neg.	neg.	neg.	+	—	neg.
57	0·912%	+	+	neg.	neg	++	neg.	neg.

Comment.—One of the latest cases of the series. Intravenous dextrose-saline was given throughout in the hope of mitigating liver damage, and extract of suprarenal cortex was given when circulatory failure appeared; both measures failed to achieve their objective. The fall of serum sodium at 3½ hours indicated the need for further investigation; the later fall during toxæmia suggested that functional impairment of the suprarenal cortex might be present without histopathological changes. Liver insufficiency was probably the main cause of death. In view of the extreme degree of liver damage, the long period of apparent well-being and the late onset of toxic signs were noteworthy.

Chart 8. Case 8.



Case 8.—**Very extensive and deep burns. Very severe shock; mild acute toxæmia; severe sepsis and pyæmia; duodenal ulcers; death on 11th day.**

E. S., female, 1 year 5 months, admitted 1½ hours after injury. Extent comparable with that in *Cases* 3 and 7. Clinical course illustrated by *Chart* 8.

POINTS.—(1) Very severe shock, probably initial merging into secondary. Capillary blood concentrated during low pressure. (2) Acute toxæmia of mild degree on 3rd and 4th days. (3) Severe bacterial infection from 7th day; hæmolytic streptococci in blood-cultures on 9th day.

Case 8.

BACTERIOLOGY: CULTURES.

Time	Blood. Aerobic	Blood. Anaerobic
6th Day.	Sterile.	Sterile.
9th Day.	Haemolytic streptococci.	
Post-mortem	Haemolytic streptococci.	

BLOOD CHEMISTRY.

Time.	CO_2 vols. %	Cl mgm. %
66 hours.	61	535
5th Day.	45	505
7th Day.	54	—
10th Day.	66	525

POST-MORTEM EXAMINATION. — Hæmolytic streptococci in burned area and blood. Pyæmic abscesses in lungs. In all organs the usual changes found in pyæmia.

Liver.—No change beyond those usual in bacterial infection.

Duodenum.—Two acute ulcers on posterior wall of first part.

Hypothalamus.—Toxic changes, but no greater than in other parts of brain.

Comment.—One of the earliest cases of the series, and incompletely investigated. It illustrated the association of very deep burns with severe shock, but with only mild acute toxæmia, and also with severe sepsis. This was a typical example of the results of hæmolytic streptococcal infection and pyæmia. There were no special histopathological changes in suprarenal glands or hypothalamus to suggest a cause for the duodenal ulcers.

Case 9.—**Scalds of moderate extent. Early onset of cerebral disturbance; death at 5½ hours; infiltration of liver with eosinophil cells.**

G. B., male, 1 year 7 months, previously healthy, sustained scalds of about 15 per cent of body surface. Picric acid was applied. At 2 hours he became apathetic and stuporose, epileptiform convulsions began and recurred at short intervals. On admission to hospital at 5½ hours he was deeply comatose and died in a few minutes.

POST-MORTEM EXAMINATION.—

Meninges and Brain, Lungs, Thoracic Lymph-glands.—All greatly congested.

Liver.—Eosinophil leucocyte aggregations, which have already been described.

Injured Area.—Hyperæmia of cutis vera, but no cellular reaction. Extreme hyperæmia and œdema of subcutaneous tissues.

Other Organs.—Nothing of importance.

Blood-culture.—No growth.

Portions of brain, lungs, and liver were tested for *picric acid* with negative results.

Comment.—A case with very unusual features, especially the rapidly fatal illness and the eosinophil-cell infiltration of the liver. The absence of picric acid in the organs examined did not support, though it did not exclude, the possibility of picric acid poisoning in a susceptible individual. Explanations such as local loss of plasma need scarcely be considered in view of the moderate extent of injury, and bacterial infection can be excluded.

Case 10.—**Extensive scalds. Treatment at first by application of gentian violet alone; acute toxæmia developed, controlled by tannic acid.**

E. R., male, 4 years 6 months, admitted thirty minutes after scalds of 20 per cent of body surface; 1 per cent gentian violet applied at intervals. At 8 hours vomiting of altered blood began and the blood-pressure fell. At 11 hours 20 per cent tannic acid applied. Toxic signs receded and had disappeared by 20 hours.

Capillary blood concentration and blood-chlorides remained unaltered throughout.

Case 11.—**Extensive scalds. Increased blood concentration without systemic disturbance.**

J. W., male, 6 years 9 months, admitted at 2 hours after scalds of 20 per cent of body surface. Secondary shock developed during local treatment, but disappeared after intravenous gum saline. Afterwards no systemic upset. *Yet the capillary blood hæmoglobin remained between* 112 *per cent and* 130 *per cent up to* 67 *hours.*

Case 12.—**Extensive burns. Hæmolytic streptococci in cultures from burned area without systemic disturbance.**

T. W., male, 4 years 10 months, admitted at 8 hours after burns of 20 per cent of body surface. Secondary shock following local treatment disappeared by 18 hours. Afterwards there was no systemic upset. *Yet hæmolytic streptococci were grown in all cultures from two areas between* 24 *hours and* 7 *days.*

Case 13.—**Extensive burns. Mild secondary shock. Severe acute toxæmia with recovery on administration of extract of suprarenal cortex.**

E. F., male, 49 years, admitted at 1½ hours after burns of 25 per cent of body surface. Severe acute toxæmia between 32 and 93 hours. Blood examination is shown in *Table* 2.

Table II. Case 13.

Time	CO_2 vols. %	P mgm. %	NaCl mgm. %	Sugar mgm. %	Urea N. mgm. %	Serum Ca mgm. %	Plasma Protein gms. % Alb.	Glob.	Van den Bergh. D.	Ind.	Fe mgm. %	Hb gm. % from Fe values	Remarks.
29 hrs	55	3·5	487	87	---	---	---	---	---	---	60	17·2	Before Toxæmia
37 "	58	4·0	440	80	63	9·6	3·62	2·10	–	+	67	19·1	During Toxæmia
53 "	66	3·8	450	84	74	---	3·00	2·80	–	+	63	17·9	
80 "	69	3·9	452	86	70	9·7	3·14	2·88	–	–	51	14·7	
101 "	68	3·7	440	84	40	9·7	3·50	2·40	–	+	50	14·3	After Toxæmia
6th Day	70	3·8	450	87	38	9·8	3·39	2·67	–	–	38	10·9	
14th "	69	3·6	452	83	37	9·6	3·44	2·50	–	–	29	8·3	

Comment.—This case is quoted to illustrate a detailed examination of blood chemistry before, during, and after acute toxæmia. It was one of the few cases in which the venous blood became concentrated (*see* values of iron and hæmoglobin). There was also a moderate rise of urea-nitrogen during acute toxæmia. The indirect van den Bergh reaction, indicating latent jaundice, was present as early as 37 hours. Otherwise there was no change of note.

DISCUSSION

The points which seem to call for consideration are the nature of shock, the classification of the clinical course, the causation of acute toxæmia, and the therapy of burns. To avoid discursive and speculative discussion, we shall direct attention mainly to features on which evidence can be offered.

SHOCK

On the first point—that is, the nature, causation, and classification of shock—we have no evidence that is at all decisive, and the most that can be said for our views is that they are favoured by clinical experience in the present and also previous studies. We shall consider first the occurrence, incidence, and aetiology of initial shock. One great difficulty in deciding this question arises from the fact that very few burned individuals can be examined immediately or very shortly after injury; few arrive at hospital within an hour of injury. It is, however, reasonably certain that a shocked condition, of nervous origin, may arise very shortly after injury; it is a highly probable mechanism of causing death, for example, in those who succumb very rapidly after burns involving almost the entire body surface. In burns of lesser extent, shock at an early stage is infrequent, rarely serious, and responds rapidly to administration of warmth and sedatives; these features it shares with known neurogenic shock arising under other circumstances—for example, during

operations on the upper abdomen.[22] Though initial shock, assuming its occurrence and nervous origin, is usually transient, there is good reason to infer that it may, for lack of adequate and timely therapeutic measures, be so prolonged and aggravated as to merge into secondary shock.

To distinguish between initial and secondary shock would also seem justifiable. Secondary shock is delayed; the fall of arterial pressure may not begin till 3 or 4 hours have elapsed. It may appear after initial shock has passed off, and it develops and progresses under conditions of warmth and relief from pain to which initial shock readily responds. The high incidence, indeed the almost invariable occurrence, of secondary shock in extensive lesions, the progressive nature of the circulatory collapse, and the absence of capacity for spontaneous recovery are other broad distinguishing features. Secondary shock, in fact, is the common and dangerous form of shock after burns. Investigation of the condition of secondary shock and of its response to treatment, to which further reference is made, does not yet permit of any decision as to its aetiology. Each of the suggested causes of secondary shock, viz., local loss of fluid, nociceptive nerve-impulses, and toxin absorption, can find support in some observations; none of them can at present be excluded. To argue in favour of, or against, one or more, or to assume that all three are operative, would be profitless.

CLASSIFICATION OF THE CLINICAL COURSE

The stages of shock having been dealt with, we pass to acute toxæmia. It has been classified, as in previous publications,[30, 31] as a condition separate from secondary shock on the one hand, and from septic or bacteriogenic toxæmia on the other. The separation can be amply justified. Though certain features, such as those associated with circulatory failure, are common to both secondary shock and acute toxæmia, and though on rare occasions differentiation may be difficult or impossible (*see Case* 5), yet in most respects the distinction is clear. Acute toxæmia is of later onset; it never begins before 6 hours, usually not till 12 hours, and occasionally serious symptoms are delayed till 40 or even 56 hours after injury. The rectal temperature is usually high in acute toxæmia, subnormal in secondary shock. A fall in blood-pressure is the central feature of shock, but is only occasional during most of the toxic phase; commonly the normal systolic level is maintained till a very late stage. The occurrence of toxæmia is much more erratic and uncertain than that of secondary shock; it may follow lesions of small or moderate extent after which secondary shock is rarely seen. There seems little justification, too, for regarding acute toxæmia as secondary shock which has been delayed or modified by treatment; the characteristic toxæmic signs may appear, and at the usual time, when local treatment has been of a primitive type or omitted altogether. In all these facts, however, there is nothing to exclude the possibility that both conditions may be produced by a similar mechanism—for example, absorption of toxic principles from damaged tissues.

There is little doubt that acute toxæmia is a condition distinct from septic or bacteriogenic toxæmia. This fact is in no way affected by the difficulty of differentiating between the conditions in certain individual cases; the difficulty arises particularly in the low-grade forms of acute toxæmia, and is partly due to the fact that we do not yet know how long non-fatal acute toxæmia or its sequelæ, such as liver damage, may continue to exert their effects.

CAUSATION OF ACUTE TOXÆMIA

We have been able with reasonable certainty to rule out all the suggested causes of acute toxæmia except one. Evidence from purely clinical sources could scarcely be more emphatic. Under the conditions of treatment, increased concentration of the blood, hæmolytic streptococcal infection of the burned area, hypochloræmia, and acidosis were inconstant features, even during the most intense toxæmia, and were evidently of no aetiological significance. Moreover, one or more might be present in cases without obvious toxæmia. Investigation is still too limited to permit of conclusions regarding changes in serum sodium and potassium, which may yield evidence of functional impairment of the suprarenal cortex. Apart from these there is no important change in blood chemistry during toxæmia, and certainly none which in itself could produce the striking toxic manifestations.

We do not, however, ignore the possibility that, under other conditions of treatment, one or more of these factors may play an important part in producing symptoms; for example, treatment which permits of continued exudation of fluid at the site of injury may be followed by serious anhydræmia, and hæmolytic streptococcal infection of the burned area may occur at an early stage. Nevertheless, neither can be regarded as a real cause of acute toxæmia. It seems to us that recently the importance of these factors has been unduly stressed, and to the exclusion of the essential cause of acute toxæmia.

It is necessary in considering the causation of acute toxæmia to take into account not only the clinical picture of overwhelming poisoning, but also the almost invariable and entirely characteristic pathological changes. The damage to the liver cells is incomparably more severe and extensive than that produced by anoxæmia, by circulatory failure, or by any of the common bacterial toxins; it is in fact an easily recognizable and specific lesion of the toxæmia of burns. In view of this lesion, it is extremely doubtful whether the hypotheses of anhydræmia, streptococcal infection, and hypochloræmia could be considered, even had we not already excluded them with certainty. The only remaining suggested hypothesis—that of the action of a toxic principle which has been absorbed from the burned area—is, on the contrary, adequate to explain both clinical and pathological features. Indeed it is difficult to suggest any mechanism other than the action of a toxin which could produce the extreme degree of liver damage. Such grounds nevertheless would be insufficient for acceptance of the toxin hypothesis were it not that evidence from animal experiment is also favourable. It has recently been shown[33] that toxin formation occurs in burned areas, and that it is the result, probably, not of bacterial action, but of autolysis of injured tissue. Admittedly the final link in the chain of evidence is still missing, namely, the demonstration of the toxin in the circulating blood during acute toxæmia. At present the available evidence is strongly in favour of the view that acute toxæmia of burns is caused by the action of a specific toxin, of non-bacterial origin, which has been absorbed from the burned area.

A few final points relating to acute toxæmia deserve mention. Measured by histopathological standards, the lesions in the liver are usually much more intense than those in other organs. There is here a suggestion either that the action of the toxin is selective or that it becomes concentrated in the liver; the latter possibility must be considered in any attempt to demonstrate the toxin in the circulating blood. Secondly, it is obvious that the liver lesion is of serious prognostic import.

Since, as is well known, extreme degrees of liver damage can be incurred before signs of insufficiency are apparent, acute toxæmia may be masked and the onset of serious symptoms delayed if other mechanisms, such as the circulation, are not especially incapacitated or are maintained by therapy. Liver function tests may therefore prove of diagnostic and prognostic value.

Lastly, acute toxæmia is clearly a complex condition, involving grave derangement of many vital functions. Although investigation of blood chemistry, so far as it has gone, has revealed suprisingly few changes, and those of minor degree only, it is not permissible to assume that tissue chemistry is similarly unaffected. The disturbance of gastric secretion, which occurs even in favourably progressing cases, suggests the possibility of other alterations as yet unsuspected.

TREATMENT

In our opinion the treatment of an extensive burn is a problem which constitutes a challenge to the best therapeutic capacity and resources at present available. Knowledge and interest, as well as energy and care, are essential if lives are to be saved. The present results of treatment would probably be improved if these injuries were universally regarded as emergency conditions which yield to none in urgency and in their demands for the highest surgical skill and judgement.

The methods of treatment, already described, are directed at the three main dangers, namely, secondary shock, acute toxæmia, and bacterial infection. In practice they have proved on the whole adequate, and at present we know of no substantial alteration which promises better results. They give a reasonable chance of recovery to the victims of burns which involve less than 50 per cent of the body surface, and a possible chance even to some with burns of greater extent. The advantages and limitations of the present treatment can best be described in respect of stages.

Initial Shock.—Our experience of severe initial shock, for reasons already stated, has been very limited, and no opportunities have arisen, for example, in cases with enormously extensive injuries for the trial of methods employed for severe secondary shock, or, in fact, of any active treatment. It is virtually certain, however, that there is a limit to the capacity for recovery; the victims of burns which implicate almost the total skin surface will inevitably die at some stage, and it is questionable whether survival to a later stage is desirable.

Secondary Shock.—Treatment by immediate coagulation of the burned area *combined with* intravenous gum-saline or blood has been used in a large number of cases during the past five years and has proved uniformly efficacious in controlling secondary shock—a pleasing contrast to the treatment of traumatic secondary shock. Evidently the combination of immediate coagulation and intravenous therapy is necessary, as has been repeatedly demonstrated when one or other has been omitted. Apparently similar effects are obtained when rapid coagulation is induced by the silver-nitrate–tannic-acid combination.[16] In only 2 of our patients did the blood-pressure fail to regain a normal level, and yet both lived till long after the time when secondary shock usually proves fatal. Both had injuries of 70 per cent or more of the body surface, and in lesions of such extent we are probably approaching the limits for successful treatment of secondary shock.

Secondary shock is certainly a common cause of death after burns in methods of treatment other than those we have described. Our inquiries on this point

allow no other conclusion. Immediate coagulation of the burned area may therefore be claimed as a life-saving measure at this stage, and we regard its use as imperative. The question will be referred to again in discussing acute toxæmia.

Acute Toxæmia.—The practical points to be discussed are the efficacy of tannic acid treatment, its mode of action, the methods of its application, and the effects of adjuvant measures. It may be accepted that the coagulation treatment of burns is still the method of choice; its many advantages enormously outweigh any disadvantages. Most observers are agreed also that it has effected a considerable reduction in mortality. On this account we felt that the use of control cases, in which tannic acid was omitted, would have been unjustifiable, although some information might have been gained. The reduction in mortality is not, however, a universal experience even when the method is efficiently employed. Seeger[24] reported no significant difference in total mortality between a series treated by tannic acid and a comparable series treated by other methods. There was, however, evidence that with tannic acid treatment the death-rate was lower during the first 5 days, thus suggesting that the tannic acid method was superior to others in combating secondary shock and acute toxæmia. In our experience[20] tannic acid undoubtedly minimizes acute toxæmia.

Davidson[9] introduced tannic acid because of its property of precipitating protein, with the object of fixing any toxic agent which might be formed in burned tissues. It has since been suggested that the benefits of tannic acid treatment are to be referred rather to the action of the protective coagulum in diminishing exudation of fluid at the injured surface, in relieving pain, or in preventing bacterial growth. If, however, our views as to the aetiology of acute toxæmia are correct, it is reasonable to assume that precipitation of the proteins of damaged cells by tannic acid minimizes autolytic changes, and thus lessens the chances of toxin formation and absorption. Davidson's original assumption as to the action of tannic acid would therefore seem to be justified.

It is quite evident, however, that tannic acid does not always control acute toxæmia, and the reason is difficult to find. The most suggestive fact in this connection is the association of acute toxæmia with certain types of injury, and therefore, presumably, with lesions of a certain depth, and, up to certain limits, irrespective of extent. As has been mentioned, very deep or very superficial injuries are often associated with mild or no toxæmia. On the other hand very extensive burns, whatever the depth, are always followed by toxæmia. We may be permitted some speculation on these observations. In deep burns, which penetrate to subcutaneous tissues, the opportunities for absorption during the first few days are probably small compared with those in burns which do not destroy the vascular dermis; it is possible, however, that in deep burns the opportunities are enhanced at a later stage by the growth of new vessels when dead tissues are becoming separated. It is at least conceivable that the depth of injury which is optimum for the development of acute toxæmia is that which is too deep to allow penetration of tannic acid to many damaged cells and yet permits of absorption from numerous intact vessels. In very extensive injuries, whatever the depth, the total mass of damaged tissue which is in relation to intact vessels and which escapes coagulation by tannic acid is bound to be great, and is probably always sufficient to produce severe toxæmia.

On the above reasoning it would seem important to secure penetration of the tannic acid. It has been asserted[17] that strong solutions—for example, stronger than 5 per cent—do not penetrate so well as weaker solutions, and are therefore less effective in controlling toxæmia. This view is not in accord with our experience. Certainly the incidence of severe acute toxæmia would seem to be no higher with 20 per cent tannic acid than with 2·5 per cent. In the last 100 consecutive cases in children treated by one of us from an early stage with 20 per cent tannic acid, there were 4 cases of severe toxæmia, of which 2 proved fatal. In a previous investigation[29] 98 consecutive cases in children, admitted before the toxic phase, were treated with 2·5 per cent tannic acid. Again there were 4 cases of severe toxæmia, of which 1 proved fatal. There is no evidence here of a significant failure to control toxæmia on the part of 20 per cent tannic acid in comparison with the weaker solution. We have felt impelled to continue its employment in preference to weaker solutions because of its undoubted life-saving properties in the treatment of secondary shock.

As adjuvant measures to the treatment of acute toxæmia we have tried intravenous administration of normal saline, gum saline, and dextrose saline, and, recently, extract of suprarenal cortex. We have seen little benefit from intravenous administration of large quantities of normal saline during toxæmia. 'Forcing' fluids is neither necessary nor advisable except in the case of frequent vomiting. Attention may also be drawn to the danger of pulmonary œdema, especially in lesions of the respiratory tract, and this risk is considerably enhanced by intravenous infusions, especially prolonged continuous-drip infusions. On general principles it would seem advisable to use a colloidal solution, such as gum saline, during the circulatory failure of acute toxæmia, when, presumably, capillary permeability is abnormally increased; particularly in the late stages the effects are, however, very evanescent. Dextrose saline has been given in the hope of mitigating liver damage, and in 2 cases in which a continuous-drip infusion of dextrose saline was used throughout the course, the liver lesion was found to be slight, though the progress of circulatory failure had been unaffected. In another (*Case* 7), however, in spite of dextrose the liver damage was extreme and was probably the immediate cause of death. Sodium bicarbonate was used in 1 case only, without special benefit.

A short report of the use of extract of suprarenal cortex in acute toxæmia has already been published[32]; 3 cases, one an example of dramatic benefit (*Case* 6), another in which given at a very late stage the extract was without effect (*Case* 7), and a third (*Case* 13) are recorded in this paper. Other personal experience is limited to 3 further cases. In 2, of low-grade slowly developing toxæmia in adults, it was without obvious action. In the other, a child with the typical circulatory failure of acute toxæmia at 23 hours, the benefit was certain. From other sources we have learned of 3 cases of 'collapse' between 6 and 30 hours after injury which responded satisfactorily to extract therapy. Our conclusions, necessarily guarded at present, are that in a proportion of cases of acute toxæmia, notably in children with circulatory failure, suprarenal cortical extract confers definite or even striking benefit. It produces no obvious change in septic burns or other severe bacterial infections. Until more information is forthcoming, we prefer not to speculate as to its action.

The necessity for continued administration of the extract over a long period has been demonstrated. Apparently, in the form of watery extract at present

available, it is very rapidly destroyed or eliminated.[5] Our present practice in extensive burns is to institute continuous-drip intravenous infusion of dextrose saline from the time of local treatment up to about 60 hours after injury. If circulatory failure develops, suprarenal cortex extract is introduced into the infusion fluid; a constant supply of extract together with sodium chloride is thus maintained. At present the only hope of mitigating liver damage seems to lie in dextrose, though success is far from assured. An agent, such as an antitoxic serum, which will neutralize the toxic principle is the obvious need of the future.

Septic Toxæmia.—The main problems relating to sepsis arise in deep burns which provide conditions exceptionally favourable to the growth of organisms—for instance, a mass of dead and damaged tissue, and impairment or abrogation of the protective bactericidal functions of the skin.

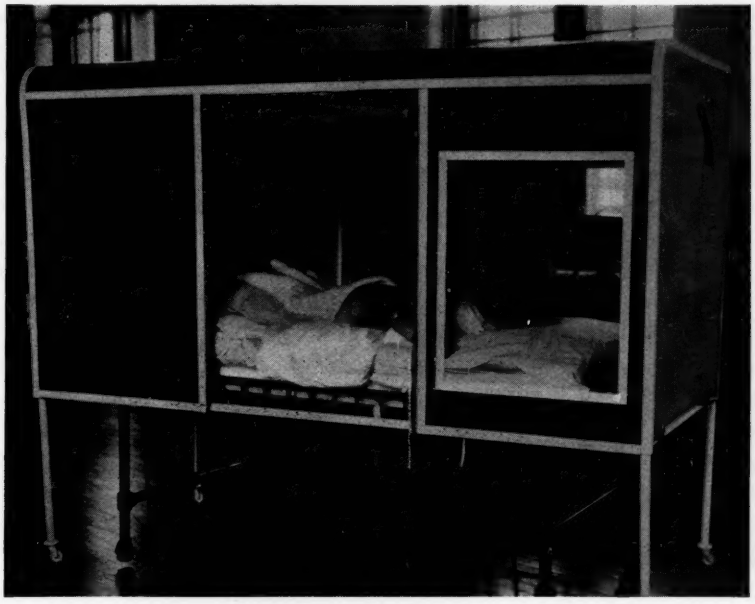


FIG. 618.—Cage used in treatment of burns. Heat supplied by morganite elements; thermostatic control; ventilation by electric fan.

The question has often been raised whether tannic acid is a suitable treatment for deep burns, on account of the frequency with which suppuration occurs under the tanned layer. The introduction of gentian violet by Aldrich[1] as a substitute for tannic acid was an attempt to reduce the incidence of infection, especially by the common invader, the hæmolytic streptococcus, on which the dye was believed to exert a selective action. Our view is that the incidence of gross infection after tannic acid has been exaggerated, owing partly to a common misapprehension—the thick inspissated yellow exudate which ultimately intervenes between living and necrotic tissues is often erroneously accepted as evidence of active suppuration. We would conclude also that the method is not in itself conducive to sepsis in deep burns. The aim is to provide a dry impermeable protective covering and so to produce and preserve conditions which are unfavourable to bacterial growth and activity, and the aim can often be achieved if the treatment is well conducted. The

alternative method of combating infection, by antiseptic wet dressings and baths from an early stage, has obvious disadvantages. The methods must be judged from considerations other than infection alone.

The additional application of antiseptics such as gentian violet and acriflavine is of great value in tannic acid treatment. Infection most commonly arises from failure to keep the coagulum dry by exposure to the air. Exposure involves the use of a heating cage, with facilities for regulating temperature and ventilation. Small cages are seldom, if ever, satisfactory. The cage illustrated encloses the cot or bed (*Fig.* 618); heat is supplied by morganite elements thermostatically controlled, and ventilation is regulated by an electric fan.

The future outlook for the prevention and treatment of hæmolytic streptococcal infection in deep burns has been rendered promising by the advent of an effective chemotherapeutic agent. We have used prontonsil or other sulphonamide preparation during the past year; it has been administered by mouth from the third day as a prophylactic measure, and supplementary doses have been given intramuscularly at the first sign of infection. The results have been distinctly encouraging, though experience is still too limited to permit of analysis.

Healing.—Since this stage has not entered the scope of the investigation in any particular respect, its treatment needs no discussion here.

Complications.—Lesions of the *respiratory tract* are likely to remain a difficult problem. The possibility of infection, particularly streptococcal, of such lesions should be borne in mind and appropriate chemotherapeusis adopted. A moist, warm atmosphere gives relief to the discomfort associated with scorching of the upper air-passages, and pulmonary lesions require the prompt and continued administration of oxygen. The prevention of other complications, such as *jaundice* and *duodenal ulcer*, probably depends upon the eradication of acute toxæmia.

Although our knowledge of the clinical course and pathology of burns is still incomplete, and the development of effective methods of treatment slow, yet progress in both respects has been steady during the past 12 years. It seems to us that the most urgent need at present, and towards which future investigation should be directed, is the complete prevention or control of acute toxæmia.

SUMMARY

1. An investigation of the clinical course of burns has been carried out in a specially selected series of 65 cases of severe and extensive injuries. The pathology of burns has been studied in 33 fatal cases, in 20 of which the clinical course had also been investigated.

2. The clinical course has been classified in respect of stages; the changes in each stage have been described in detail.

3. The important feature of the investigation relates to the causation of the stage of acute toxæmia. Such factors as anhydræmia, changes in blood chemistry, and bacterial infection have been excluded as primary or essential aetiological agencies. The older hypothesis of a specific toxin, which is formed in burned tissue and absorbed into the blood-stream, receives support from this investigation. The important and characteristic pathological lesion of acute toxæmia is degeneration and necrosis of the liver cells. An occasional, and apparently hitherto unrecognized, change is infiltration of the liver, and sometimes of other organs, by eosinophil leucocytes.

4. Evidence for the main conclusions is detailed in a description of illustrative cases.

5. The application of the results to the treatment of burns is discussed.

This work owes its inception and inspiration to the late Dr. J. S. Haldane. We are indebted for clinical facilities to the Surgeons of the Royal Hospital for Sick Children, the Royal Infirmary, and the Western General Hospital, Edinburgh, and for pathological material to the staff of the Pathology Department, Royal Infirmary, Edinburgh.

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OSTEOCLASTOMA OF THE AXIS VERTEBRA

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OSTEOCLASTOMA is one of the commonest tumours affecting bone; Geschickter and Copeland[1] analysed a series of 1040 bone tumours and found 214 cases of osteoclastoma, this condition being second in the list in order of frequency. The diagnosis of this tumour in the long bones is generally fairly simple on account of its position in the epiphysis and its characteristic radiological appearance, but when occurring in unusual sites such as the spine the clinical diagnosis presents considerable difficulty. That these tumours do occur in the spine is shown in 26 cases, details of which have been collected from the literature. Lewis[2] in 1924 collected 17 proved cases (including a case of his own), Santos[3] reported on 6 more cases in 1930; Milch[4] in 1934 published a case of giant-cell tumour of the spinous process of the 3rd cervical vertebra, and further cases have been recorded by Lindsay and Crosby,[5] and Camurati[6].

Analysis of these cases shows that osteoclastoma may arise in any part of the vertebral column, the lumbar region being the commonest site, whilst the cervical region was involved in 9 cases. The occurrence of the tumour is not confined to any particular area of the vertebra, and the previously reported cases show that it may be situated in the vertebral body, spinous process, transverse process, or lamina, the body and the spinous process being the areas most frequently involved. Those involving the spinous process present less difficulty from the point of view of diagnosis, as there is usually a palpable tumour; and on account of their accessibility they are the most favourable for surgical treatment.

The clinical diagnosis of osteoclastoma of the vertebral body is beset with considerable difficulty. It has to be differentiated from tuberculosis of the spine, the commonest affection involving the vertebral bodies. In this latter condition there is usually definite 'knuckling', and the radiograph shows contraction and irregularity of the intervertebral space, with collapse of the vertebræ affected, combined frequently with a shadow cast by a paravertebral abscess. In osteoclastoma there may be some collapse of the vertebral body due to the fact that its structure is weakened by the tumour and its consequent inability to support the superimposed weight of the body; this collapse is, however, a secondary effect, and not the important feature seen in tuberculous affections. This is well seen in the radiograph shown in *Fig.* 619.

The diagnosis of secondary metastases of the spine from osteoclastoma may be difficult, as the former do not present any typical X-ray appearances. Brailsford[7] points out that some cases show rarefaction, whilst others exhibit evidence of osteosclerosis, and in order to confirm the diagnosis careful search must be made for the primary growth.

Chronic osteomyelitis of the spine is often associated with septic foci elsewhere, and the patient may be febrile, whilst the X-ray picture with its new-bone formation is quite characteristic. Hæmangioma of a vertebral body may simulate osteoclastoma, but in the former condition the reticulation seen in the radiograph is much finer, and there may be signs of compression myelitis.

CASE REPORT

HISTORY.—P. W., male, aged 45, a gardener, had always been a particularly healthy man until twelve months before admission, when he slipped and felt pain in the neck, which lasted for three weeks and was worse at night. The symptoms then disappeared for seven months, when he slipped again; the pain in the neck recurred and was much more severe than previously, and persisted until admission to hospital.

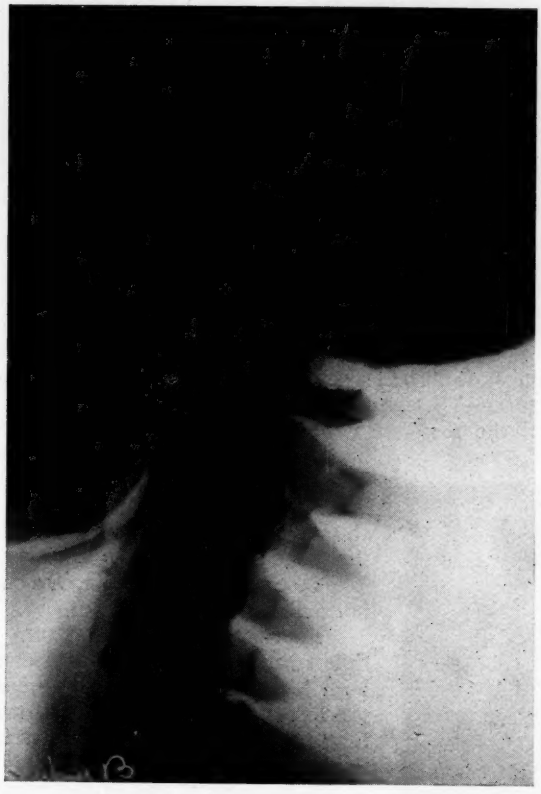

FIG. 619.—Lateral radiograph of cervical spine showing osteoclastoma of the axis.

ON EXAMINATION.—He was a healthy-looking man. There was prominence of the 2nd cervical spine, with pain and limitation of movement in all directions. He stated that the right side of the face and neck felt numb, but there was no anæsthesia or other sensory changes. There was no evidence of retropharyngeal or other abscess formation.

TREATMENT AND SUBSEQUENT PROGRESS.—The patient was treated on a Jones frame with head-piece. There was no relief of his symptoms, and the severe pain at the back of the neck, which at times radiated over both occipital regions along the course of the great occipital and 3rd occipital nerves, caused the patient much distress. Morphia was prescribed frequently to enable the patient to get some relief. Three months after admission he developed œdema of the left leg and pain over the femoral vessels in the groin, due to thrombosis of the left femoral vein. This was followed a fortnight later by a small pulmonary infarct at the right

base, with pain, pleural rub, and slight staining of the sputum. This, together with the thrombosis, cleared up. He remained eighteen months in hospital, his condition gradually deteriorating, with almost continuous severe pain in the neck and occipital region, which was aggravated by any movement. There was never any loss of motor power or sphincter

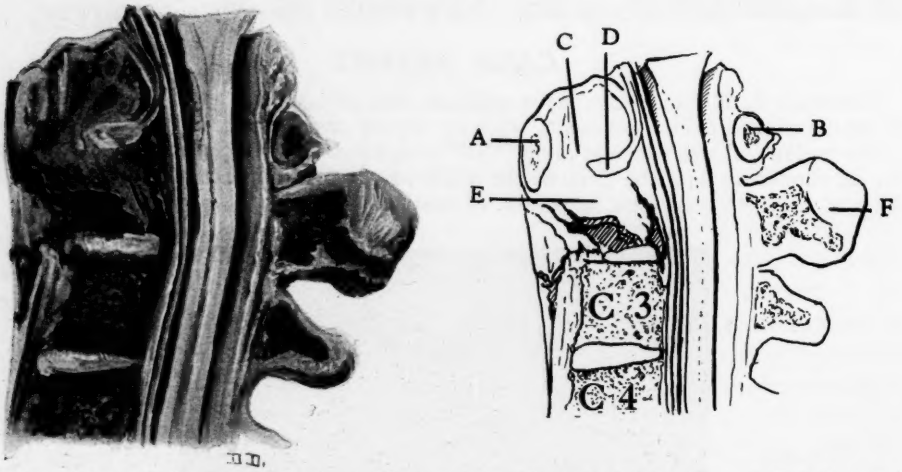

FIG. 620.—Showing appearance of upper four cervical vertebræ on sagittal section. A, Anterior arch of atlas; B, Posterior arch of atlas; C, Neck of odontoid process; D, Transverse ligament; E, Tumour tissue in position of body of axis; F, Spine of axis.

disturbance. Six weeks before death he developed a cough with profuse foul-smelling purulent sputum, with moist sounds scattered over both lungs, and later dullness at the left base with absent breath-sounds, death being due to multiple pyæmic lung abscesses with empyema.

X-RAY EXAMINATION.—A lateral radiograph of the cervical spine shows prominence of the spinous process of the axis due to backward displacement (*Fig.* 619). The vertebral body is compressed, and presents a vacuolated appearance with irregular coarse bony trabeculæ; the anterior border of the body is not outlined, and a few bony strands project forwards beyond the normal outline of the bone; there is also involvement of the lamina. The intervertebral spaces above and below the axis are not delineated.

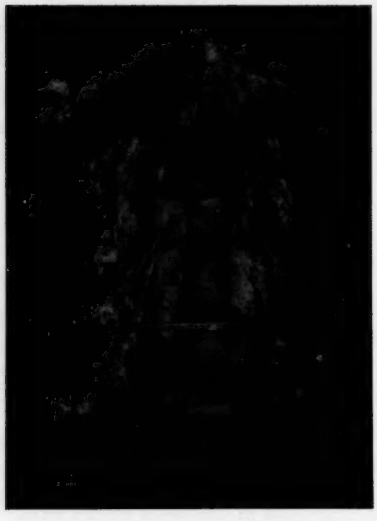

FIG. 621.—Anterior view of specimen, showing erosion of body of axis.

POST-MORTEM EXAMINATION.—This revealed multiple small pyæmic lung abscesses scattered throughout both lungs, the largest being the size of a marble, and the left pleura was full of foul-smelling pus. The left lung near the base showed a superficial area of necrotic tissue, suggesting that the pus in the pleura was due to the bursting of one of the numerous lung abscesses. The cervical spine was removed *in toto* for hardening and section. On stripping the soft parts from the front of the spine prior to removal there was erosion of the front of the body of the axis, and a drachm of dark hæmorrhagic fluid escaped from a cavity in the body of the vertebra, which suggested that the lesion might possibly be a hæmangioma, especially as the blood exuded under pressure.[8] (*Fig.* 621.)

PATHOLOGICAL ANATOMY (Professor S. L. Baker).—*Macroscopic.*—The main naked-eye features are well seen in the illustration and key diagram (*Fig.* 620), which show the specimen cut in sagittal section in the position of the upper four cervical vertebræ. The tumour involves mainly the axis, the body of which is almost completely

destroyed, and is represented by a mass of friable red and brownish-red tissue (E). The odontoid process (the neck of which is in the position marked C) is also largely replaced, and forms a rounded mass covered in part by a thin shell of bone and retained in place by the anterior arch of the atlas (A) and the transverse ligament (D). The cartilages of the odonto-atlantal joint surfaces are also preserved, and can be seen in the illustration as a vertical pale streak behind the anterior arch of the atlas. The superior and inferior articular surfaces of the axis are intact, but the cancellous tissue of both the laminæ is invaded. In addition to the destruction of the axis, the lesion extends on to the upper part of the anterior and posterior surfaces of the third cervical vertebra; there is some invasion of the anterior common ligament and muscle attachments in this region. There is slight involvement of the cancellous tissue of the anterior arch of the atlas, but this is only visible microscopically. There has been no invasion of the posterior occipito-axial ligament or of the spinal dura mater over the lesion.

Microscopical.—The lesion has resulted from an osteoclastoma which has involved mainly the body, odontoid process, and part of the lamina of the axis. Tumour tissue characteristic

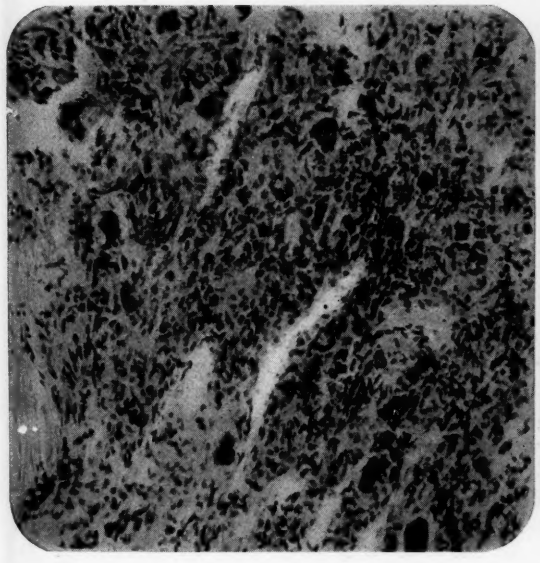

FIG. 622.—Low-power view of tissue from region of body of axis, showing osteoclasts lying in a tissue composed of spindle cells. (× 175.)

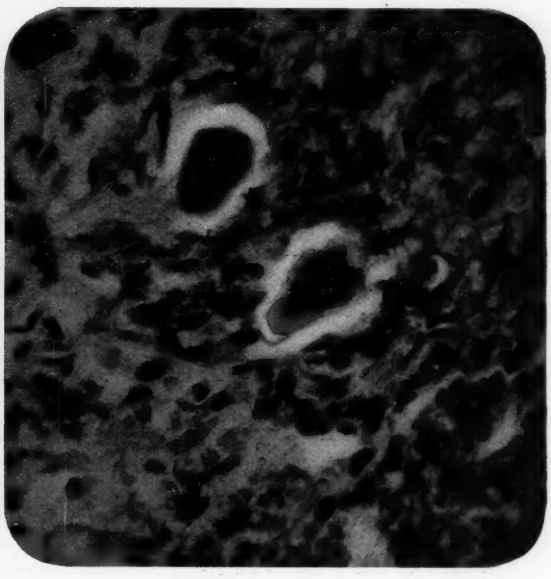

FIG. 623.—High-power view (stain Weigert's iron hæmatoxylin and van Gieson's method) showing darkly stained collagenous tissue extending into osteoclastoma. (× 500.)

of an osteoclastoma is found in certain situations, particularly in the red tissue which occupies the position of the body of the axis. In other places osteoclasts are few or absent, and varying amounts of collagen fibre are present.

The following range of appearances is therefore seen: (1) Typical osteoclastoma, i.e., non-collagenous tissue composed of spindle cells with deeply staining oval nuclei and containing numerous multinucleated osteoclasts (*Fig.* 622). (2) A similar spindle-cell tissue without osteoclasts; (3) A spindle-cell tissue containing numerous collagen fibres, the cells being more sparsely scattered than in (2) (*Fig.* 623); (4) A very vascular finely reticular connective tissue composed of cells with light staining oval nuclei and showing numerous interlacing fine collagen fibres. Abundant thin-walled blood-vessels are found throughout, being particularly obvious in the last-mentioned tissue. These various types of tissue merge one into the other, so that although the last-mentioned bears no resemblance to an osteoclastoma, a series of connecting links suggest that it is the result of a fibrotic healing process in such a tumour. This reticular tissue is well developed in the rounded mass which has replaced the upper part of the odontoid process. This mass shows a shell of bone composed

in part of lamellar bone and in part of trabeculæ of newly-formed woven bone which extend into the spindle-cell reticular tissue (*Fig.* 624).

A section through the anterior arch of the atlas shows for the most part normal cancellous bone, the spaces of which are occupied by blood-forming marrow, but at one spot near the lower border the marrow space contains a small area of collagenous reticular tissue similar to that described above.

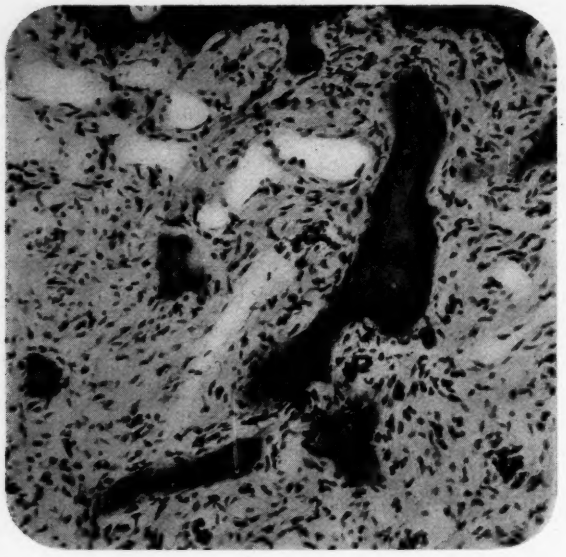

FIG. 624.—Low-power view, showing newly-formed trabeculæ of woven bone extending into fine connective tissue in the odontoid process. (× 120.)

Comment.—The histological appearances in this case appear to correspond with that seen in some of the cases reported by Lewis.[2] This writer stresses the relative frequency of healing with new-bone formation in osteoclastomas of the spine. In the present case much of the tumour has undergone fibrosis, but there has been only a small amount of new-bone formation. Lewis remarks that the tendency to spontaneous healing raises some doubt as to the neoplastic nature of the process in many of these cases. In the present instance the 'expansion' of the odontoid process and the invasion of ligamentous and muscular tissue on the anterior surface of the affected vertebræ can leave little doubt as to the neoplastic nature of the process.

DISCUSSION

This case emphasizes the difficulty of the clinical diagnosis in osteoclastoma of a vertebral body. There were certain features resembling Pott's disease; the radiograph showed compression of the vertebral body, with obliteration of the intervertebral space above and below the lesion, but reticulation with vacuolation of the actual body is not a feature of tuberculosis of the spine. The fact that treatment by rest did not have the slightest effect in relieving the severe pain, which became gradually worse, was definitely against a diagnosis of Pott's disease, as systematic rest almost invariably relieves the pain in this condition, and relief is obtained quite early in the treatment. There was no evidence of abscess formation externally or internally, although the coughing up of large quantities of pus just before death suggested the possibility of a retropharyngeal abscess having burst into the air-passages.

Reticulation of the vertebral body is characteristic of hæmangioma, but in this case the reticulation was much coarser than is usually seen in hæmangioma, and there were no parallel vertical striations which are characteristic of the latter condition.[9] A provisional diagnosis of hæmangioma was made at autopsy in view of the appreciable amount of blood which exuded under pressure from the vertebral body on stripping its anterior surface. This finding suggests the possibility of other similar cases being designated hæmangioma in error unless a complete microscopic investigation is made.

The persistence of severe pain throughout the whole course of the disease was very suggestive of neoplasm.

We wish to express our thanks to Mr. T. P. McMurray, who suggested the publication of this article, for his advice and criticisms.

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A CASE OF CIRSOID ANEURYSM OF THE SCALP

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T. H., male, aged 45 years, lorry driver, admitted December 18, 1936.

History.—In 1914 a small soft swelling was first noticed on the right side of the head, and this had gradually increased in size since that time. The lump was quite painless until ten days ago when, in attempting to stand upright, he struck his head against an iron girder in the roof of his lorry. Following this, the swelling was painful for five or six days and began to grow more rapidly. He had never suffered from headaches or felt any throbbing in the head. There was never any discoloration of the skin noticed, or bleeding from the lump. When aspirated by his doctor after the recent injury, however, bright blood was withdrawn. He had had no treatment, his eyesight was normal, and there were no other symptoms.

Physical Examination.—Growth of hair on the scalp was thinner in the part involved by the tumour. After shaving, an irregular lump, roughly triangular in shape, was to be seen occupying the right temporal and frontal regions. The border of the tumour corresponding to the base of the triangle was 2 in. long and just transgressed the midline. The apex was directed laterally and forwards, and became continuous with the tortuous and very prominent superficial temporal artery (*Fig.* 625). In its longest transverse diameter the lump measured 2½ in. Its surface was raised about ½ in. above the general level of the scalp, but just behind the middle of the tumour it projected 1 in. above the general level, and again near the apex its surface was elevated nearly 1 in. The postero-lateral border of the tumour followed roughly the line of the fronto-parietal suture, and about its middle was a small prolongation of the tumour along the posterior auricular vessels.

Close scrutiny of the shaven scalp failed to reveal any sign of recent or old trauma, and the colour of the skin overlying the tumour was normal. The whole swelling was seen and felt to pulsate synchronously with the pulsation in the superficial temporal artery. On palpation the lump was extremely soft and easily compressible, but on releasing the pressure it immediately returned in a symmetrical fashion to its original size; lowering the head and straining seemed slightly to increase its prominence. The peculiar softness of the tumour gave an impression to the examining fingers similar to that of a depressed fracture.

Four groups of vessels were seen running into the tumour. The right superficial temporal artery was principally involved. Its course was plainly outlined and appeared to be a very tortuous one. As can be seen from the drawing, the artery approached the tumour by a very circuitous pathway. The wall of the vessel was soft, and when its lumen was obliterated by digital pressure, pulsation in the tumour promptly ceased, it diminished slightly in size, and refilled only very slowly when emptied. Other vessels to assist in supplying the lump were the right posterior auricular, the right supra-orbital, and the left superficial temporal arteries, the course of the last-named being barely discernible. Obliteration of these vessels individually or collectively failed to affect the pulsation of the lump, nor was it

rendered more tense by these procedures. A loud systolic bruit was heard over all parts of the swelling, but not when the superficial temporal artery was occluded.

Pulsation in both common carotid arteries was equal in force. There was no cardiac hypertrophy detectable clinically, nor evidence of any valvular disease. The blood-pressure was 115/90 mm. Hg.

The patient was a strong man, of good colour and nutrition, and examination of the other systems revealed no evidence of abnormality.

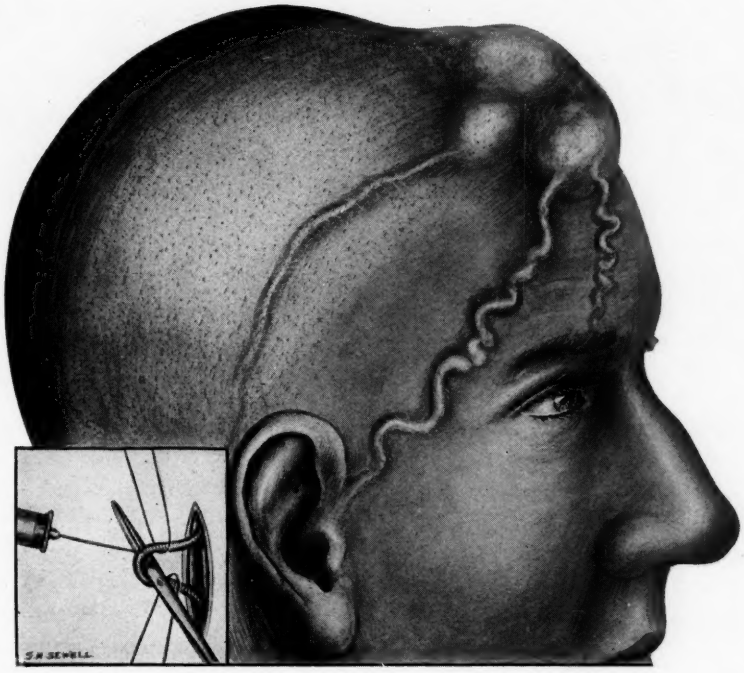

FIG. 625.—Drawing of patient, showing the situation of the tumour and its vessels of supply. The superficial temporal artery is seen to be principally involved, and the irregularity of the surface of the tumour is well shown.

The *inset* shows the thorotrast injection being made into the superficial temporal artery. The redundant loop of artery prolapsed from the wound in the manner shown, indicating the great lengthening and tortuosity of its walls.

X-RAY EXAMINATION.—In the preliminary radiograph, just anterior to the bregma the tumour can be seen standing out in profile as a plateau-like elevation of the soft tissues (*Fig.* 626). The underlying bone shows no depression or absorption. Actually, the tumour was found at operation to involve only the skin and subcutaneous tissues, and emissary connections were few in number.

Under local anæsthesia the superficial temporal artery was exposed through a small vertical incision overlying the zygomatic arch. The vessel was so elongated and tortuous that, on incising the covering fascia, it projected about ½ in. from the wound in the form of a redundant loop. Despite its prominence clinically, the calibre of the vessel was but little increased. A correspondingly fine needle and a 20-c.c. Record syringe were now used to inject 18 c.c. of thorotrast into the distal end of the exposed vessel (*see Fig.* 625, *inset*). The injection was made as follows: 8 c.c. were first given as rapidly as possible; and the tumour was then emptied

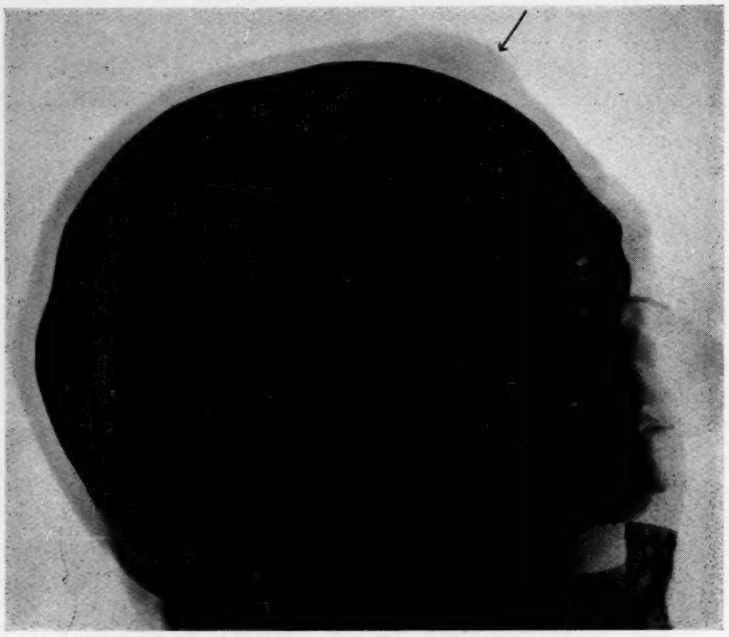

FIG. 626.—Preliminary radiograph, showing the tumour in profile.

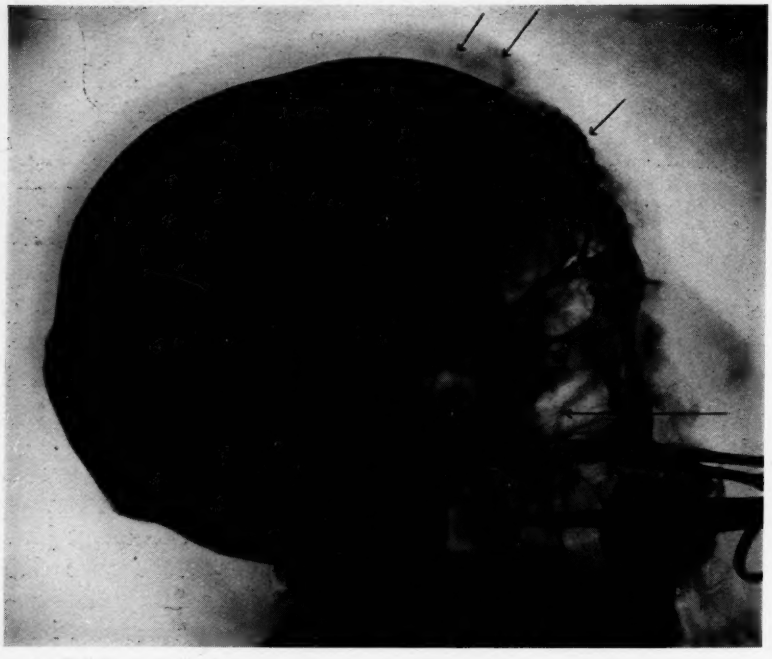

FIG. 627.—Radiograph taken immediately after thorotrast injection, showing opaque fluid in the tumour. The sigmoid sinus and bulb of the internal jugular vein are seen outlined.

by manual pressure. After an interval of fifteen seconds the remainder of the thorotrast was given, the pressure on the tumour immediately removed, and a rapid succession of radiograms was taken. The total time that elapsed between the commencement of the injections and the final picture was about thirty seconds.

In the second plate (*Fig.* 627) the needle point is seen lying in the lumen of the superficial temporal artery, which has been outlined by the thorotrast. Its wavy outline is plainly seen running up towards the tumour. The latter stands out in relief superficial to the surface of the skull. On close inspection it is shown to be constituted by a plexus of convoluted and dilated vascular channels. The efferent veins are not visualized, but a phlebogram of the sigmoid sinus and the bulb of the internal jugular vein are plainly seen, showing that a considerable quantity of thorotrast found its way indirectly into the cerebral sinuses.

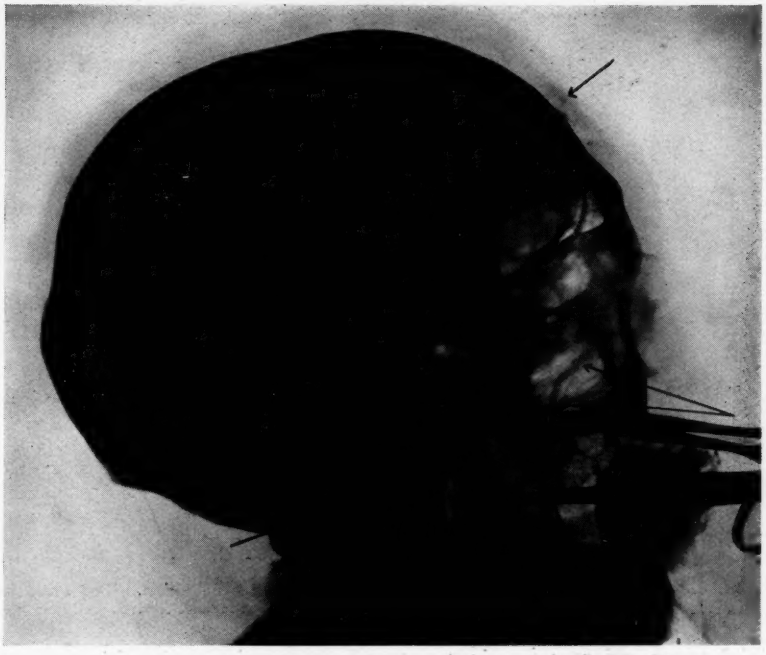

FIG. 628.—Final radiograph taken 30 seconds after injection. The sigmoid sinus and bulb of the internal jugular vein are seen outlined.

The third plate (*Fig.* 628), taken between one and two seconds later, shows that the thorotrast has been completely swept through the tumour. The bulb of the jugular vein and the sigmoid sinus are plainly outlined as before.

The artery was ligated on either side of the needle puncture, and the wound closed with a silkworm-gut suture.

OPERATION.—After an interval of six hours, the tissues of the scalp were infiltrated with ½ per cent novocain, and an incision was made down to the pericranium, outlining an omega-shaped flap, which included the tumour and had its base just above the level of the zygoma. Hæmorrhage was effectively prevented by digital compression of the tissues on either side as the incision was lengthened, the galea being turned back with artery forceps in the usual manner. The flap was quickly

raised and the tissues of its base compressed with a curved intestinal clamp, in the manner recommended by Searby. Opposite the site of the tumour a few emissary vessels necessitated the application of forceps, and the surface of the skull was found to be slightly depressed over a circular area of about 1½ in. in diameter.

The tumour was now very much reduced in size, but its limits were still quite recognizable, and it was dissected off the deep surface of the flap. It consisted of a plexiform bundle of thin-walled vessels, which were situated in the skin and subcutaneous tissue. Great care was required to avoid 'button-holing' the skin while yet removing the tumour completely from it. The 'feeding' vessels were clearly recognizable as its periphery was cleared, and each was carefully dissected out to the corresponding edge of the flap, those at its base being secured. A portion

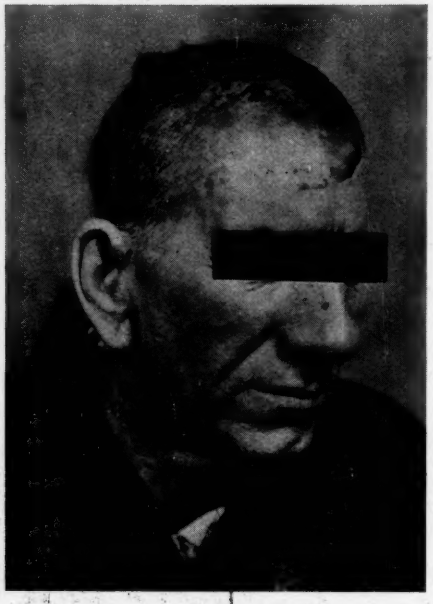

FIG. 629.—Photograph of patient taken twelve days after operation. The tumour has entirely disappeared, and the skin incision is well healed.

of superficial temporal artery was preserved for section. At this stage the clamp was removed, but only a few small bleeding-points required ligatures. The galea and skin were sutured, a small rubber tube being inserted into the subaponeurotic space at the base of the flap. The operation was completed in forty minutes.

SUBSEQUENT PROGRESS.—The drainage-tube was removed on the second day, and the stitches on the tenth. There was some slight necrosis of the edge of the flap at one point, but the wound was healed when the patient left hospital on the fifteenth day. *Fig.* 629 shows the condition on the twelfth post-operative day.

HISTOPATHOLOGY.—

1. *Superficial Temporal Artery* (*Fig.* 630).—The whole of the muscular tissue of the media is abnormal and has undergone hyaline degeneration, with a great diminution in the number of nuclei, which are shrunken. There is much increase in the amount of internuclear material. The intimal coat is irregularly thickened, in many cases to a gross degree.

2. *Tumour.*—A section taken from one part of the tumour shows a vein recently thrombosed; the thrombus has organized to a considerable extent, there being a marked fibroblastic reaction (*Fig.* 631). The appearance suggests a traumatic, rather than an inflammatory, lesion.

FIG. 630.—Section of superficial temporal artery.

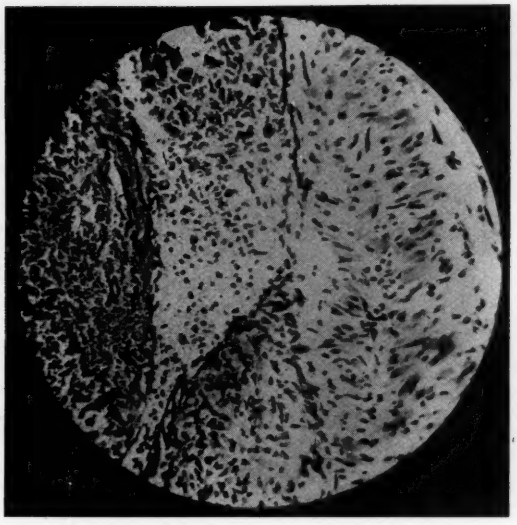

FIG. 631.—Section from one part of tumour, showing thrombosis in a large vein.

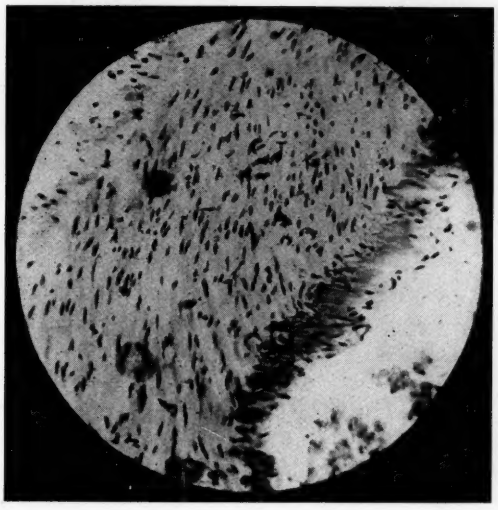

FIG. 632.—Another section of the tumour, showing a greatly dilated artery in section. There are marked changes in the structure of its wall.

At another part of the tumour an artery is seen in section, and here the same medial and intimal changes as previously noted in the superficial temporal artery are present, but to a much greater degree (*Fig.* 632). There is considerable

vacuolation of the medial coat, indicating lipoid change; this is present also in the intima. The whole artery is grossly dilated. The media shows focal degeneration.

Conclusions.—Though the pathology of cirsoid aneurysm is obscure, it seems justifiable to draw the following conclusions from a study of this case:—

1. Since the thorotrast can be seen in only one dilated and tortuous vessel running through the tumour, the latter probably rapidly communicates with many large veins in which the medium is too diluted to throw a shadow. The likelihood of this conclusion is increased by the excellence of the phebogram of the sigmoid sinus and bulb. Moreover, sections from different parts of the tumour showed the majority of the vessels to be of the nature of veins.

2. That the disease process involves not only veins but also arteries is proven by the dilated, tortuous, and histologically abnormal character, not only of the artery sectioned in the tumour, but also of the superficial temporal vessel at a distance from it.

I am very much indebted to Mr. E. Rock Carling for permission to publish details of this case. I wish to thank Dr. Kerley for his great assistance with the radiological study of the tumour, and Dr. Allchin for his help with the prints. Dr. R. J. V. Pulvertaft kindly furnished the histological report and microphotographs.

EXPERIMENTAL SURGERY

AN EXPERIMENTAL INVESTIGATION INTO THE CAUSE OF PARALYSIS FOLLOWING SPINAL ANÆSTHESIA*

By A. D. MACDONALD and K. H. WATKINS

One of the authors has investigated a series of cases in which paralysis in the form of a cauda equina syndrome followed the employment of spinal anæsthesia in man (Ferguson and Watkins[5]). It appeared from a study of this series that the complication was especially liable to follow the employment of a solution known as duracaine, although occasional cases have been recorded in the literature after the use of other agents. It seemed certain that the lesion resulted from some toxic effect of the injected drug rather than from the mechanical trauma of the needle. The composition of duracaine ('heavy') was:—

Procaine HCl, 'Planocaine' brand ..	0·1 g. per c.c.
Gliadin	0·00325 g. per c.c.
Glycerin	0·1 g. per c.c.

In 15 per cent ethyl alcohol.

This was later altered to the following:—

Procaine HCl, 'Planocaine' brand ..	0·1 g. per c.c.
Gum acacia	0·002 g. per c.c.
Glycerin	0·0416 g. per c.c.

In 15 per cent ethyl alcohol.

As it was uncertain which of the constituents of duracaine was responsible for the toxic effect, the matter was subjected to laboratory investigation, the results of which are recorded below.

While the actual anæsthetic agent seemed the most probable cause of the undesired symptoms, it must be remembered that intrathecal injections of stronger solutions of alcohol have been used for the production of prolonged anæsthesia, while glycerin is not without possible action on nervous tissue.

METHOD OF EXPERIMENT

The animal employed in this investigation was the cat. This animal had been extensively utilized by one of the authors (A. D. M.[1]) for other experimental work on spinal anæsthesia, and appeared well suited to the present task. It was evident that in order to conduct this research on a practical scale, some means must be taken to render the incidence of paralysis considerably greater than that occurring in man (less than 1 per cent). It appeared to be reasonable to bring this about by the injection of the drug in much greater relative bulk than that employed in man. Usually the concentration injected was the same as that of the particular constituent

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of ' heavy ' duracaine under examination, except that the alcohol and glycerin were also given in even greater concentration.

Each animal was given light ether anæsthesia after an injection of atropine sulphate, gr. $\frac{1}{200}$. A fine needle was introduced between the laminæ of the sixth and seventh lumbar vertebræ, and after obtaining a good flow of cerebrospinal fluid, 0·5 c.c. of the particular fluid was injected. This dose is about five times the usual clinical dose, calculated on equivalents in terms of body weight. If this dose is exceeded, respiratory depression may follow, the drug affecting not only the intercostal but also the phrenic roots. With a dose of 0·5 c.c. the tendon-jerks in the forelimb are not as a rule noticeably affected. In introducing the needle, great care is taken to minimize trauma. In particular, the needle is not pushed too far, lest it puncture the venous sinuses behind the bodies of the vertebræ. Unless a free flow of cerebrospinal fluid is obtained, experience has shown that the injection is likely to be a failure. Similarly, if the fluid contains blood, the resulting anæsthesia may be less certain in its duration and extent.

The cat was held vertically or fixed in a reclining position with the head uppermost. This was intended to limit the spread of the anæsthetic and sustain a good concentration in contact with the lumbar roots for some time. When the injection contained procaine, the knee-jerks were observed to disappear quickly, and as the cat emerged from the ether anæsthesia, paralysis of the hind limbs and hind parts of the body was manifest in its attempt to move about. When the solution contained only alcohol and/or glycerin, some degree of weakness was sometimes observed when the animal first recovered from the ether, but the knee-jerks never disappeared.

Lesion Produced.—The animals were kept under observation for varying periods following the injection. In a certain number, as shown below, a typical cauda equina syndrome ensued. Thus there was paralysis of the bladder, sphincter ani, and tail; sometimes weakness of the hind limbs, and also impairment of sensation. In those animals which developed paralysis, the lesion was always manifest when the animal was examined one to three days after injection.

The lesion was not always as extensive as that described above, and frequently the paralysis was limited to the tail. In such cases, when the animal was stroked, the base of the tail was elevated whilst the remaining two-thirds or three-fourths hung flaccid from the base. Sometimes also, the tail would deviate to one or other side. The distal part of the tail also was insensitive to pinching and pressure.

In the complete lesion, the picture was identical with that observed by one of the authors following division of the roots of the cauda equina or excision of the conus medullaris in dogs (Watkins[6]). The bladder became very greatly distended, and by firm pressure urine could be expressed in a stream which ceased instantly on release of the pressure. In an animal which was not paralysed, the bladder was never particularly large and expression was impossible. On rare occasions compression would set up urination in a normal animal, but under such circumstances the stream continued after release of the pressure until by reflex micturition the bladder had emptied. After about three weeks the affected bladder became very much reduced in size, but it continued to be unusually large and its contents expressible in the manner above described. The contents of the bladder in one cat, which was kept alive for six months after its spinal anæsthesia, remained easily expressible, though at the last examination the stream produced by expression did not seem to stop quite so abruptly on release of the pressure. In another cat, expression began

to be more difficult after six weeks, but the bladder was still abnormally distended after seven months.

The paralysis of the anal sphincter showed itself by abnormal prominence of the anus, and frequently it would be seen to be widely relaxed over a scybalous mass. Impairment of sensation in the tail and in the area surrounding the anus was suggested by the extremely dirty condition of these areas in previously clean animals; and the tail was shown to be insensitive to pinching and deep pressure. Flaccidity of the tail was demonstrated in the manner above described. In the two animals which were kept alive for a prolonged period, the tail began to show gradual recovery after six or seven weeks and could be raised to the vertical after three or four months.

RESULTS

The full results may be summarized in *Table I* as follows:—

Table I.—Experimental Results

	Total Injected	Not Paralysed	Paralysis of Bladder, Anal Sphincter, and Tail	Paralysis of Tail Only	Total Paralysed	Percentage Paralysed
Alcohol and glycerin	23	23	–	–	–	–
'Heavy' duracaine	23	10	9	4	13	56
10 per cent procaine (Planocaine brand)	10	5	2	3	5	50
10 per cent procaine (Novocain brand)	23	17	4	2	6	26
5 per cent stovaine (Barker)	10	4	–	6	6	60

These results appear to be conclusive proof that the toxic agent which is responsible for the paralysis after duracaine is the procaine itself. That other anæsthetic agents besides procaine may produce paralysis is indicated by the experiments in which stovaine (Barker) was employed. It is not suggested that the above figures establish any difference between the brands of procaine.

The failure to produce a single case of paralysis following the employment of alcohol and glycerin (frequently in higher concentration than that in 'heavy' duracaine itself) serves to show that they are not directly responsible for the lesion, and still further disposes of the view that the condition results from trauma by the point of the needle. In one animal in which extensive trauma had occurred in repeated unsuccessful attempts to obtain a flow of cerebrospinal fluid, no injection was made, nor did any paralysis subsequently develop.

Further evidence is provided in the same direction by a series of experiments in which have been correlated the incidence of sequelæ with the concentration of local anæsthetic used. In a series of 20 cats injected with 0·5 c.c. of 2·5 per cent procaine no prolonged paralysis was seen. In a further 20 treated with 5 per cent

procaine 2 showed pronounced tail droop, but bladder and rectal symptoms did not arise. From the earlier table we conclude that in 56 cats injected with 10 per cent solutions sequelæ appear in over 40 per cent. A few were then given 20 per cent procaine. In the first 2, with 0·5 c.c. of solution, this dosage proved too much for respiration; in 6 others, therefore, 0·3 or 0·4 c.c. was given, according to body weight. One more died acutely, leaving 5 survivals, of which 4 developed severe lesions, affecting tail, rectum, bladder, and even the hind legs. (*See Table II.*)

Table II.—Relation of Dosage of Procaine HCl to Incidence of Symptoms

Percentage Concentration	Number of Animals	Number showing some Paralysis	Percentage Paralysis
2·5	20	0	0
5	20	2	10
10	56	24	43
20	8	4	80*

* 3 died acutely.

As in the case of man, the ultimate paralysis seemed to be localized to the terminal spinal nerves, although the injected anæsthetic agent caused a much more widespread paralysis of thirty to sixty minutes' duration. This implies that the agent only causes lasting paralysis of those nerves which are bathed by the substance in its full concentration. The nerves which it reaches after dilution with cerebro-spinal fluid recover their function rapidly—as soon as the agent is absorbed or destroyed. Because of the paralysing effect of the drug in its full concentration of 10 per cent, the injection of cats with a relatively greater bulk determined a much higher incidence of paralysis in these than in man.

It is of special interest to observe that paralysis was found to follow the use of stovaine as well as of procaine, the active constituent of duracaine. It seems certain that the incidence of paralysis following the use of stovaine in man is small, very few cases having been recorded in the literature. It is doubtful whether in this investigation 'heavy' duracaine is more damaging than the preparations of procaine alone, and the question remains as to whether or not the addition of alcohol and glycerin (innocuous in themselves) enhances the toxic action of the procaine itself.

DISCUSSION

It has long been held that the intrathecal injection of spinal anæsthetics in high concentrations leads to toxic changes in the spinal cord and spinal roots, both in animals and in man (van Lier,[3] 1907; Spielmeyer,[4] 1908; Wossidlo,[7] 1908). Spielmeyer also observed paralysis of the hind limbs and diminished tendon-jerks in a dog after the intrathecal injection of 0·04 g. of stovaine. In 1933, Lundy, Essex, and Kernohan[2] showed that the intrathecal injection of 5 c.c. of 20 per cent procaine caused extensive paralysis in dogs. These workers, however, did not find paralysis from solutions of 17·5 per cent or less, nor, surprisingly enough, was paralysis noticed in dogs injected with only 2·5 c.c. of 50 per cent solution.

It appears certain from the present investigation that the spinal anæsthetic agents in the concentrations ordinarily employed in man are toxic to nerve tissues to a degree capable of causing serious lasting paralysis in animals. In man their rapid dilution with cerebrospinal fluid is sufficient, except in rare instances, to prevent such paralysis, and in any event determines that when such paralysis does occur it is of relatively limited extent. In the experimental animal the bony canal is much more filled by nervous tissue, and there is only a small amount of cerebrospinal fluid wherewith the injection can be diluted.

It would seem reasonable to suppose that the risk of paralysis would be considerably reduced in practice if the anæsthetic agents were injected in less concentrated form. Indeed, Lundy, Essex, and Kernohan[2] state that following a temporary paralysis from the use of 10 per cent procaine in man, they " have avoided the use of solutions stronger than 7 or 8 per cent, and commonly employ concentrations of 3, 4, or 5 per cent solutions as it leaves the syringe ".

On the other hand, the volume of the solution cannot be indefinitely increased without the introduction of the risk of respiratory depression. If, however, the usual 2 to 3 c.c. of 10 per cent procaine—a dose, incidentally, which may be double the maximum recommended by the British Pharmacopœia, 1932 (150 mg.)—is first diluted with cerebrospinal fluid and injected slowly so that dilution time is available, delayed toxic symptoms should be rare.

An attempt was made to investigate the histological changes induced in the spinal roots in prolonged paralysis, but the difficulties of interpretation are such that at present no useful contribution to this aspect of the problem can be offered, though its importance is fully appreciated.

SUMMARY

Intrathecal injections of spinal anæsthetic solutions and the various constituents of these solutions into a series of about 150 cats have shown :—

1. That certain cocaine substitutes, in concentrations commonly employed clinically, though in relatively larger doses, may produce lasting paralysis comparable to lesions of the cauda equina.

2. That improvement in these cases is usually apparent in some six weeks, but cure may be incomplete in six months.

3. That the common constituents of the solution other than the local anæsthetic drug do not of themselves produce lasting paralysis in the concentrations in which they are normally employed.

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THE NERVOUS CONTROL OF GASTRIC SECRETION
AN EXPERIMENTAL STUDY

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INTRODUCTION

THIS paper forms the first part of an experimental study of the neurogenic factors involved in peptic ulceration, and is being carried out at the Buckston Browne Surgical Research Farm.

The connection between organic cerebral disease of certain types, mainly tumours in the region of the third ventricle and certain varieties of internal hydrocephalus, and peptic ulceration must now be accepted as more than coincidence. Cushing, in his Balfour lecture of 1931, described with his usual clarity the sequence of ideas on the aetiology of peptic ulceration from Rokitansky in 1841 and Virchow in 1853 up to the time of his oration. The conception of peptic ulceration as a local disease of the stomach—a conception traceable to Virchow and held by some even to-day—is quite illogical if one attempts to correlate the whole clinical picture with such an idea. The aetiology of peptic ulceration, at present uncertain, must explain and embrace all the following points: (1) Why does an ulcer appear at all? (2) Why does it remain chronic? (3) Why is there a definite ulcer-bearing area? and (4) Why does the patient with peptic ulceration present the well-known psychological 'make-up'?

The mass of experimental work on the stomach itself and clinical observations accumulated over many years have elucidated but one of these points—namely, that the passage of acid gastric contents over a pre-existing ulcer is an essential factor in chronicity; and the empirical therapeutic measures have been directed against this factor alone. The recent investigation of clinical material by Davies and Wilson (1937) has established beyond reasonable doubt the connection between psychological disturbance and the exacerbations of the local manifestation of peptic ulceration. This, coupled with the clinical observations of Cushing, Mabel Masten, and many others, and supported by the experimental work of Beattie, Keller, and their co-workers, lends support to the growing conviction that peptic ulceration is a disease in which the appearance of an abnormal gastric mucosa is but a part.

Referring to the conception of the aetiology of peptic ulceration as a local disease, Cushing states "out of this has come the highly unprofitable search for its primary cause in the walls of the stomach itself, a search beset by pitfalls and contradictions". But when one reviews the past experimental work done on the vegetative nerves to the stomach, one does not always find agreement. It is possible, however, that here these deviations can be explained by the fact that one is dealing with mixed nerves in the sense that each contains both parasympathetic and sympathetic fibres and the admixture is not constant either in pattern or proportion.

The fact that experimental neurogenic ulceration can be produced in the stomach, though so far no chronic ulceration can be made to take place, stimulated one to review and investigate the changes in gastric secretion which may be initiated, and to determine, if possible, whether conditions can be produced favourable to the chronicity of such mucosal deficiencies. The constituents of the gastric juice which have been investigated are: (1) Volume; (2) Acidity; (3) Mucus. These are the three factors which appear to be of paramount importance in the peptic ulcer patient.

The technical difficulties of chronic nerve stimulation are being attacked, but have not yet been solved, and all the series of nerve stimulation experiments to be described are acute, i.e., the animal is not permitted to recover.

The problem of chronic neurogenic ulceration is being approached at the moment, but the results are too premature for publication.

METHOD OF EXPERIMENT

The experimental work to be described was carried out entirely on cats and dogs, and the following series of experiments was performed:—

1. Chronic or recovery experiments:
   Division of the various vegetative nerves to the stomach.
2. Acute or non-recovery experiments:
   a. Splanchnic nerve stimulation;
   b. Cœliac ganglion stimulation;
   c. Vagal stimulation;
   d. Hypothalamic stimulation.

Much of the work done on this question has been performed before, but one or two interesting facts have come to light. Throughout the whole work particular attention has been paid to the acid content, the volume, and the mucus content of the gastric secretion.

Technique Employed in Collecting the Gastric Secretion.—

1. *The Recovery Experiments.*—In these the stomach tube was used, and although by this method a rough estimate only can be gauged, it was felt that in view of the mass of physiological work performed in the Pavlov and Heidenhain pouches it would be unnecessarily redundant to repeat it. In any case the primary object of these recovery experiments was concerned with ulcer production, and the investigation of the gastric secretion was carried out as a procedure of secondary importance.

2. *The Acute Experiments.*—Several series of acute experiments were carried out, mainly on cats, the experiments lasting six hours and more. Then the animals were killed.

The technique employed is as follows: The animal is kept without food, but allowed water to drink for twenty to twenty-four hours. By that time the stomach is empty of food. The cat is anæsthetized by giving intravenous chloralose in a dose of 70 mg. per kilo body-weight. The abdomen is opened to the right of the midline and the stomach delivered into the wound. In all cats the presence of hair cannot be predicted, so as a routine an incision about 1 in. long is made in the greater curvature about its middle, and all contents and debris removed. The stomach is then gently washed out, using cotton wool soaked in distilled water,

so as not to affect the acidity. The incision into the greater curvature is then closed in two layers in the usual manner. An incision is now made into the first part of the duodenum about $\frac{1}{2}$ in. from the pylorus, and a rubber tube whose diameter is roughly that of the duodenum is inserted from the duodenum through the pylorus into the stomach. About 2 in. of tube is lying in the stomach, and this has two side holes in it, the proximal one lying just inside the stomach. A stitch through

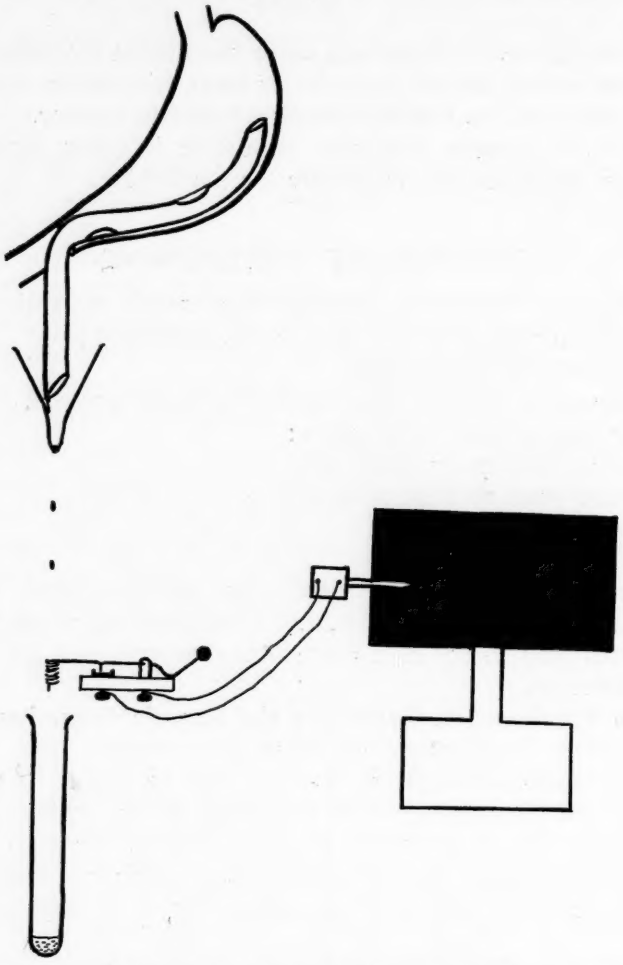

FIG. 633.—Diagrammatic representation of the method for collecting gastric secretion in the acute experiments. *See* text for explanation.

the tube secures it to the duodenum, and as there are no holes in the tube as it is passing through the duodenum, the only secretion collected is the gastric juice. The abdomen is now closed by means of 'through and through' sutures and the proximal end of the tube projects outside the body.

The animal is now placed on its face and the whole animal tilted slightly to the right by means of a block under the left side of the table top. The proximal end of the tube is laid in a filter funnel which has been drawn out to a known diameter, and this funnel is placed immediately above a Condon drop counter.

In this way the number of drops of juice formed are determined and recorded electrically on a smoked drum. The size of the drop is calculated, and varies in size from 0·045 c.c. to 0·037 c.c., according to the mucus content of the secretion. This method of collecting the gastric juice has been adopted after considerable experiment, and appears to be the most satisfactory in ensuring that all the juice secreted by the stomach is collected, and that none is either passed into the duodenum or left in the stomach itself. (*Fig.* 633.)

After this procedure the animal is allowed to settle down for one hour at least, before any attempt is made to influence the gastric flow, and by that time the steady flow for the particular animal is established, which flow varies from animal to animal. After the juice has been recorded by the drop counter it is caught in a dry sterile test-tube, which is changed each time an analysis is made. The acid content only has been investigated, and this is titrated against N/20 NaOH, using Töpfer's reagent as an indicator.

Technique Employed in Nerve Stimulation.—

Electrodes.—

1. A satisfactory 'recovery' electrode is wanting, and work is being carried out on this question with a view to constructing an electrode which can be left in position for several weeks and remain effective.

2. The electrodes used in the acute experiments have been two in number: (*a*) A fluid electrode modified after that by Collison; and (*b*) An ordinary platinum wire electrode. As only short bursts of stimulation are used in investigating the gastric reaction, the two types are of equal value, but when the nerves are stimulated for some hours, then of course the fluid electrode is used only.

Method of Producing the Nerve Stimulus.—The mode of the stimulus is two-fold. The first method employs an ordinary induction coil, which has a rocker arm in the primary circuit, by which the make-and-break can be regulated accurately from 5 per second upwards. A rhythmic stimulation is also produced by introducing a metronome into the circuit. This method gives quite good results, but its disadvantage lies in that the actual current used is too high, and in time tends to damage the structure which is being stimulated.

The second method uses a thyratron valve giving a saw-toothed discharge, with a second valve which can be used to regulate the current output. In this way a current of 0·5 to 1·0 milliampere is used, varying a little with the resistance of the structure stimulated, and a voltage of about $\frac{1}{2}$ volt, which is just subminimal for striated muscle. All rates of stimuli have been used, and it has been found that a rate of 50 per second is most potent in producing changes in the gastric secretion.

EXPERIMENTAL RESULTS

1. Recovery Experiments.—A series of recovery animals was prepared, the anæsthesia for cats being either ether or subcutaneous nembutal and for dogs morphia and nembutal, gr. $\frac{1}{4}$ of the former and gr. $\frac{1}{2}$ of the latter per kilo body-weight. One of the four following operations was performed:—

a. Bilateral splanchnicotomy;
b. Excision of the cœliac ganglia;
c. Bilateral vagotomy;
d. Bilateral splanchnicotomy plus vagotomy.

These animals were kept from periods of one to ten months before being finally killed. Prior to the operations a gastric analysis was done, and also the histamine reaction determined. It was found that the operative procedures mentioned in no way influenced the normal acidity of the gastric juice and the rate of flow permanently. Immediately after the nerve ablations those animals which had had vagotomy, particularly dogs, did have a lower normal acidity, but this quickly became normal once more within a month. On the other hand, the histamine response was as before, whether carried out immediately after or some months later (*Figs.* 634, 635). From what one now knows of the histamine response,

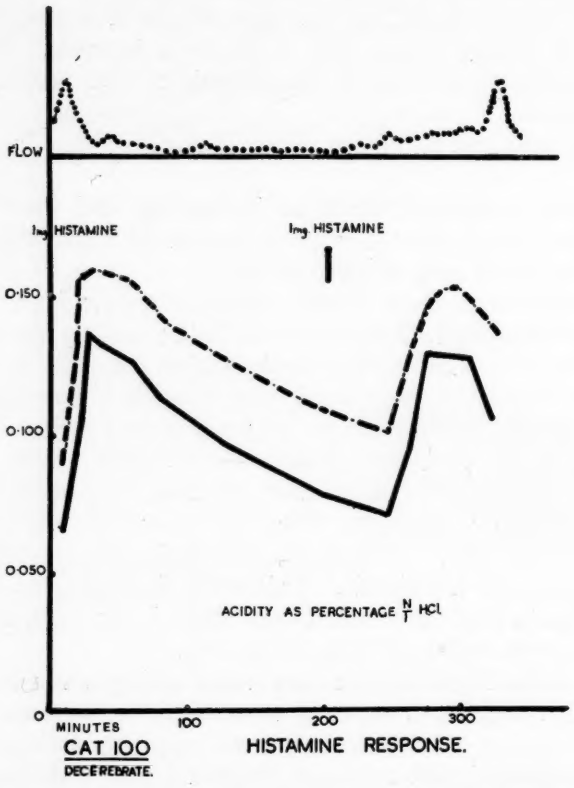

FIG. 634.—Normal histamine response. Abscissa = Time in minutes. Ordinate = Acidity expressed as percentage of N/1 acid. .....: Rate of flow of juice; -.-.-: Total acidity; ——— Free acidity.
NOTE.—All the succeeding graphs in this paper have been constructed in the same way, using the same scale and method of drawing.

its behaviour under these circumstances is to be expected. The action of histamine is directly on the parietal cell alone, through the medium of the blood-stream, and the cell is made to discharge the acid precursors contained therein. In the several series of experiments performed, whether acute or chronic, this response to histamine was not appreciably affected by any procedure on the vegetative nerves to the stomach, whether the nerves had been ablated or were being stimulated. The other cellular elements in the gastric mucosa are not activated by histamine, and its value as a test of gastric function is therefore small. As an indicator of the maximum power of the parietal cell it is extremely useful, and for this reason only

was used in these experiments. The concentration of free acid and the volume of free acid produced by histamine was very much greater than that produced by any nervous stimuli.

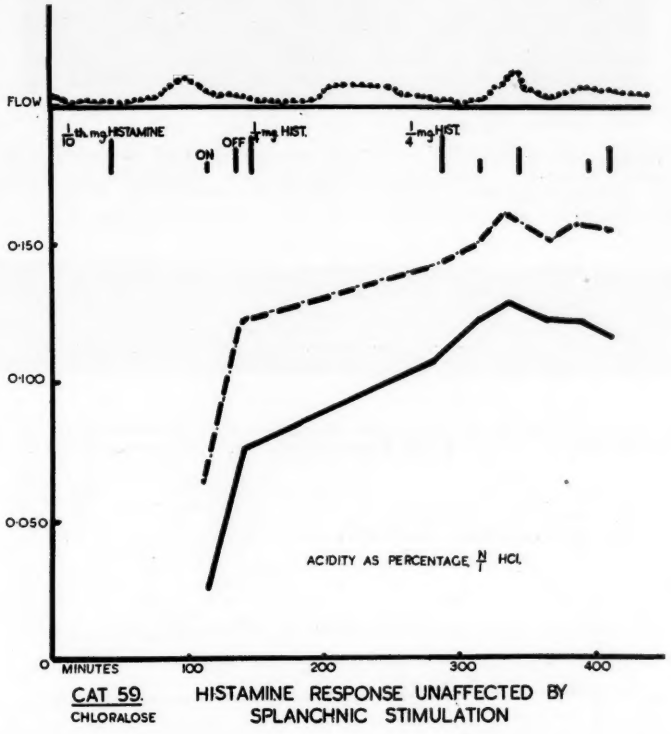

FIG. 635.—Histamine response unaffected by splanchnic stimulation. Short vertical line = Stimulus on; Long vertical line = Stimulus off. Stimulus used: Rhythmic at 15/2/50 (*see* text for explanation). Three small intramuscular injections of histamine, the middle one being allowed to act without nerve stimulation taking place. Analysis not commenced until after first injection had passed off.

2. Acute Experiments.—Several series of acute experiments were carried out, mainly on cats, the experiments lasting six hours and more. Then the animals were killed.

a. Splanchnic Nerve Stimulation (*Figs.* 636–638).—In all these acute experiments the cat was anæsthetized by intravenous chloralose in the dose of 70 mg. per kilo body-weight, preceded by ether administration. The splanchnic nerves have been stimulated both above and below the diaphragm with identical results. Technically it was more convenient to use the transthoracic approach and pick up the nerve as it neared the diaphragm. Stimulation was then carried out through an open thoracotomy, using a positive pressure method of artificial respiration. Below the diaphragm the approach was retroperitoneal, by an incision below the last rib and stimulating the nerve just before it entered the cœliac ganglion. The nerve was crushed proximal to the point stimulated to abolish the involuntary twitchings of the animal which occur when the intact nerve is used. The greater splanchnic alone and the greater plus lesser splanchnics have been stimulated with no appreciable difference in response from the gastric mucosa. The rate used was either a rhythmical stimulus at 15/2/50, i.e., fifteen stimuli every two seconds

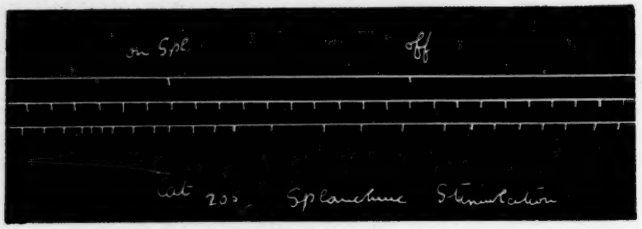

FIG. 636.—Splanchnic stimulation. Rate of flow recorded, using a three point recorder on a smoked drum. Top line = Indicator; Middle line = Minute intervals; Bottom line = Drops of gastric juice.
NOTE: All the succeeding tracings have been recorded in the same way. Stimulus used: thyratron valve at 50 per second.

FIG. 637.—Successive vagal and splanchnic stimulation using thyratron valves at 50 per second. The letter A means that the secretion was taken for analysis. The three analyses are (as percentage N/1 acid):—

		Free Acid	*Total Acid*
1.	Resting acid	0·026	0·064
2.	After vagal stimulation ..	0·092	0·120
3.	After splanchnic stimulation ..	0·067	0·103

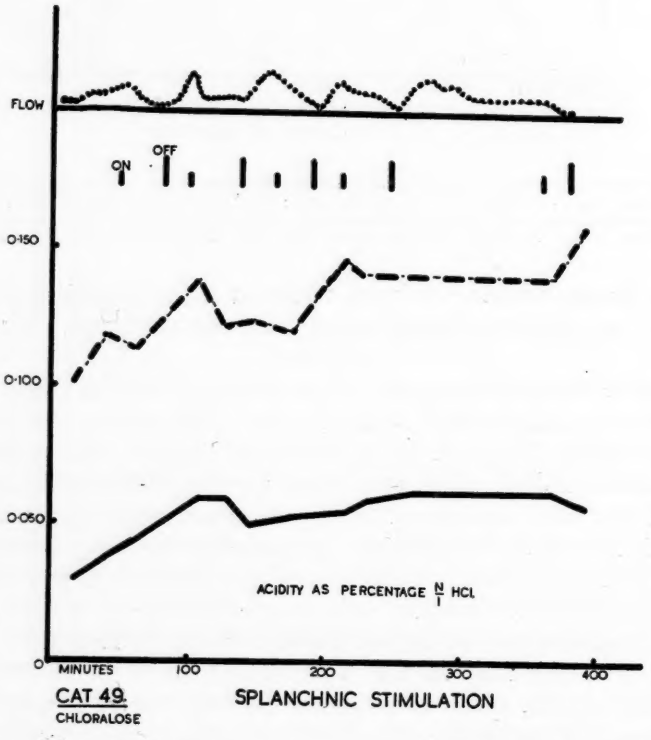

FIG. 638.—Splanchnic stimulation. Stimulus used: Rhythmic at 15/2/50. Note large amount of combined acid, which is a good indication of mucus content of the juice. Acidity slowly rising up to end of experiment.

at the rate of 50 per second, or else a continuous stimulation at the rate of 50 per second.

No artificial means were used in order to promote a flow of juice if the cat's stomach was slow in secreting. If the stomach did not begin secreting on its own, then that particular investigation was abandoned for the time being. Once a regular flow became established, the nerves were stimulated at intervals, and the same response obtained every time. After a latent period of about three to five minutes, the flow would become less, and in some experiments would fade out altogether. Then, after cessation of the stimulus, the flow returned after a latent period of a minute or two. This effect is, of course, explained most easily by a pure vaso-constriction of the gastric vessels, but in addition to an alteration in the flow of

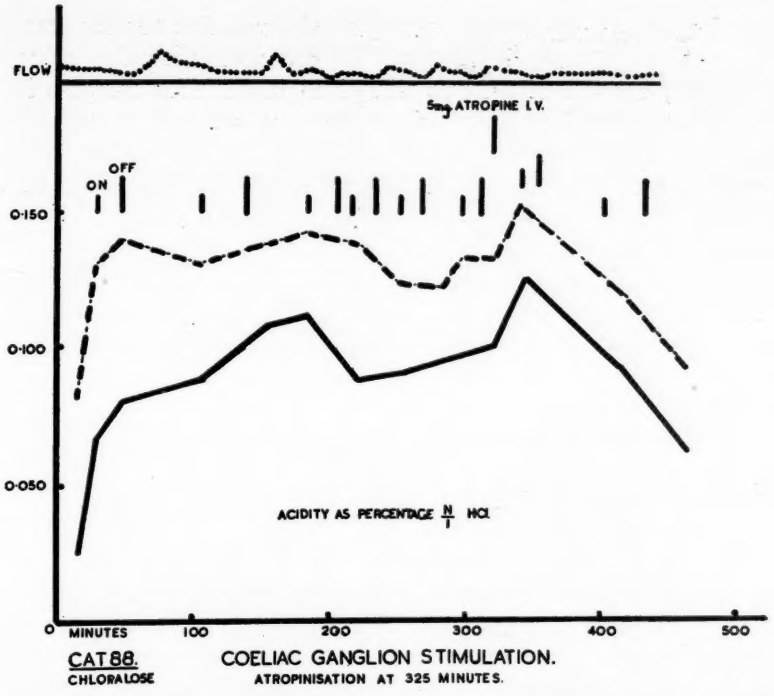

FIG. 639.—Cœliac ganglion stimulation. Stimulus used: Rhythmic at 15/2/50. Note much less combined acid than in *Fig.* 638; less marked effect on flow; steady fall of acidity after atropinization, unchecked by further cœliac ganglion stimulation.

gastric juice, the acidity invariably rose considerably above the resting acid level. This rise varied from animal to animal, but some rise in acidity was always produced.

Another phenomenon noted was the appearance of mucus in the secretion, occurring so constantly that it was quite significant. When this occurred the proportion of free acid fell and the combined acidity rose accordingly. It is impossible accurately to measure the amount of mucus secretion in the same way that a relatively mucus-free juice can be measured and recorded, and the observation must be taken on trust, but the whole question of mucus secretion will be referred to again later in this paper.

In passing, an interesting observation was noted in all these acute experiments, and this was that when the splanchnic or cœliac or vagal stimulation was stopped,

the free acid did not remain steady at the level maintained during the stimulation, but continued to rise. The impression one obtained was that the secretion of free acid depended on some sort of a trigger action, and that when any nervous stimulus was thrown in, then the acidity continued to rise until a certain level was reached, varying with each animal; and until that equilibrium had been attained it was impossible further to influence the acidity. With regard to this increased acidity caused by splanchnic stimulation, the probable explanation was that this was due to the parasympathetic content in the splanchnic nerve, and, with this in view, a series of animals was given intravenous atropine in sufficient dose to paralyse the vagal endings. In these it was found that the acidity fell sharply, and that further splanchnic stimulation was ineffectual in raising it again, but the secretion of mucus was greatly increased by the stimulation. There seems to be general agreement amongst anatomists that the splanchnic and the abdominal vagi are 'mixed' nerves in the sense that each contains sympathetic and parasympathetic nerve-fibres. Exactly where this admixture occurs is not quite certain, but it is certainly more central than the thorax, because cats who had had bilateral vagotomy just below the lung root some weeks previously, still retained a sufficient vagal content in the splanchnics to influence the acidity.

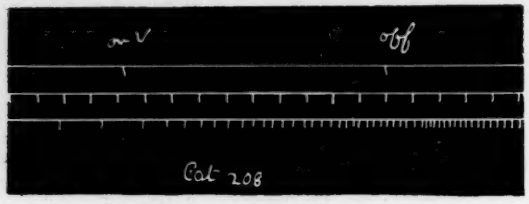

FIG. 640.—Vagal stimulation. Stimulus used: Thyratron at 50 per second.

b. Cœliac Ganglion Stimulation (*Fig.* 639).—The ganglia were approached through the same incision as that used in the lower route to the splanchnic, and stimulation was carried out retroperitoneally. A number of cats had the cœliac ganglia stimulated instead of the splanchnic nerves, and in these there were slight but definite differences. The rate of flow was not so easily influenced, and the acidity was raised on the whole to a greater degree. After atropinization, however, the behaviour of the juice was as with the splanchnic experiments; the acid fell and cœliac ganglion stimulation was more effective in depressing the flow, but

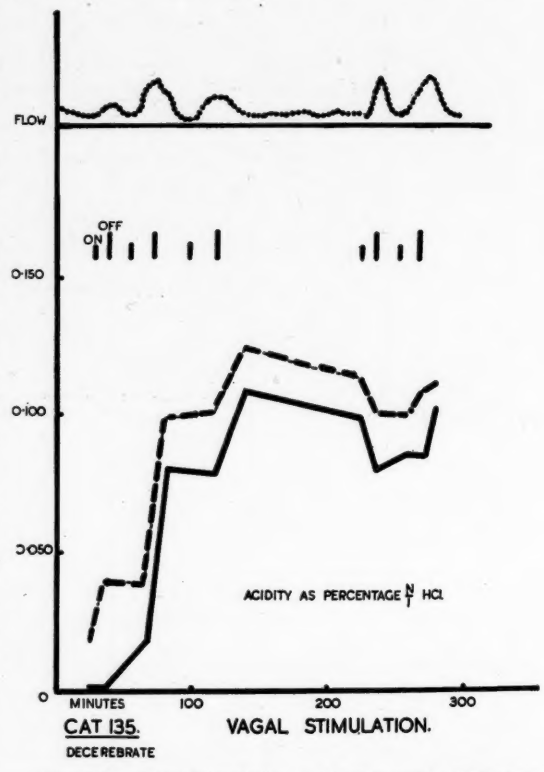

FIG. 641.—Vagal stimulation. Stimulus used: Thyratron at 50 per second. Note that the amount of combined acid is small; less than in *Figs.* 638, 639.

had no influence on the acidity. This difference is to be expected, because a definite leash of vagal fibres passes from the left or posterior vagal trunk to the cœliac ganglia, and some must find their way along the gastric vessels to the stomach.

c. Vagal Stimulation (*Figs.* 640, 641).—The vagi were stimulated through an open thoracotomy at a point below the lung route, the left or anterior trunk being usually used. This was quite a short series, as the investigation has been made already by a number of workers, and merely confirmed the well-established facts.

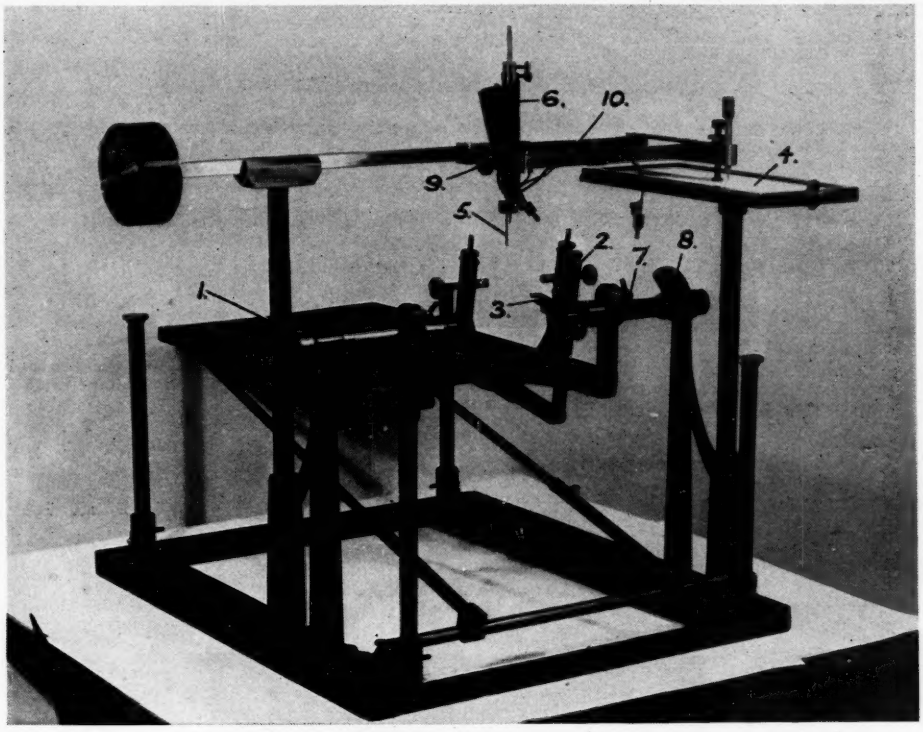

FIG. 642.—The Souttar machine. 1, Platform; 2, Headpiece; 3, Mouthpiece; 4, Chart; 5, Electrode; 6, Vernier scale; 7, Locking; 8, Protractor; 9, Screw for adjusting height of electrode; 10, Assembly for holding electrode built on pantograph system in ratio 2 : 1—i.e., 2 mm. on scale = 1 mm. at point of electrode.

As has been noted many times before, it was necessary to use a stimulation-rate of 50 per second to obtain any response. The lower rates had no apparent effect on the flow whatever. The results obtained were the same as those of a good many previous workers—after a latent period of about three to five minutes the juice began to increase in flow and acidity. The acidity obtained was, in the main, appreciably higher than that obtained by splanchnic stimulation, and tended to persist after stimulation had ceased for a considerable time. The rate of flow, too, once increased, remained at a higher level for some time before slackening off to the pre-stimulation level.

d. Hypothalamic Stimulation (*Figs.* 642–647).—For this purpose the Souttar machine modified by Beattie was used, which machine is standardized for the head

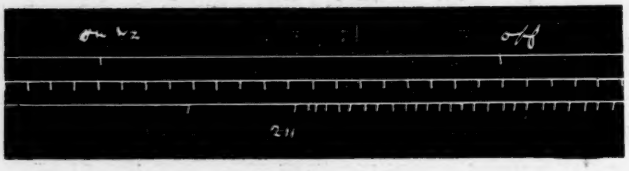

FIG. 643.—Stimulation of left anterior hypothalamus 2 mm. behind bregma, 2 mm. from midline, and 2 mm. from base line (*see* text). Stimulus: Thyratron at 50 per second.

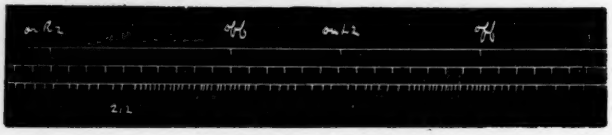

FIG. 644.—Stimulation of right and left halves of anterior hypothalamus 2 mm. behind bregma, 2 mm. from midline, 2 mm. from base line: the latent period here is shorter than usual. Stimulus: Thyratron at 50 per second.

FIG. 645.—Stimulation of right anterior hypothalamus at bregma level, 2 mm. from midline and 2 mm. from base line: a long latent period and poor response, and probably due to 'spread' backwards to the 'sensitive region.' Stimulus: Thyratron at 50 per second.

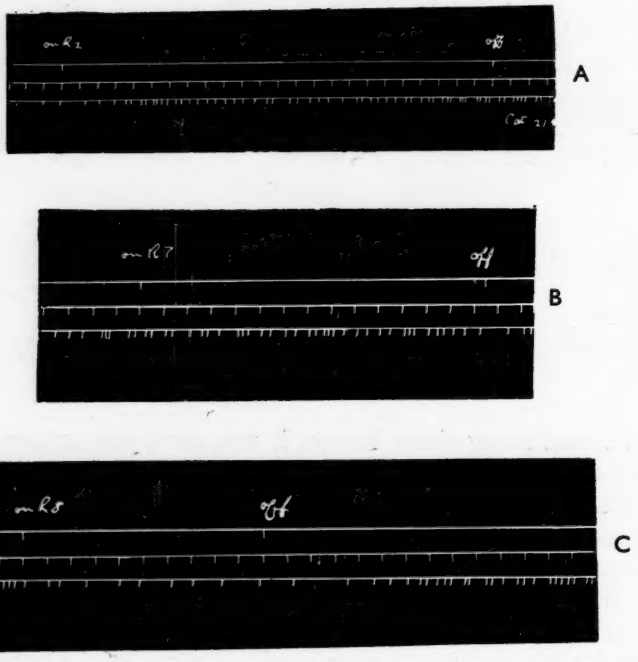

FIG. 646.—Showing the effects on one animal of stimulation at three points: A, 2 mm. behind bregma at the usual depth, and producing a parasympathetic response. B, 12 mm. behind bregma, 2 mm. from midline, and 8 mm. from base line. This has had no appreciable effect on the flow. C, 14 mm. behind bregma, 2 mm. from midline, and 8 mm. from base line. The flow has diminished, resembling the effect produced by splanchnic stimulation (*see Fig.* 636). Stimulus: Thyratron at 50 per second.

of a 2½-kilo female cat. The procedure is as follows: The cat is anæsthetized with chloralose in the usual dose of 70 mg. per kilo body-weight, and then the collecting tube is sutured into position in the stomach. The head is now fixed in the headpiece, and the metal plate which abuts on the hard palate is tilted downwards 10° from the horizontal. This ensures that the base of the brain is horizontal in relation to the machine, as it compensates for the slight projection of the incisor teeth. The scalp is incised sagittally, and the bregma defined and marked on the chart. A rectangular segment of skull is removed in the midline and the sagittal sinus is secured by Cushing silver clips before the dura is turned back. With the head in this position a fine bipolar circular electrode made from a No. 15 hypodermic needle, completely insulated except at the tip, is plunged vertically through the brain to the appropriate level.

It is assumed that all the brains used were exactly the same size, or, at any rate, that the variation was so slight that the number of experiments done cancelled out any such discrepancy. From previous work done on the hypothalamus by Beattie and his co-workers, it was assumed that the approximate surface marking of this part of the brain stretched in an antero-posterior direction from the bregma to a point 15 mm. behind that; and that the lateral boundaries of each half were the plane of the sagittal suture internally and another vertical plane 3 mm. external to that.

Within this rectangular area points were stimulated systematically at 2 mm. distance from each other, both in the horizontal and vertical planes. The depth was measured, not from the surface, but in the reverse direction from an arbitrary base line common to all the experiments. This base line was determined by passing a dummy electrode identical in dimensions with that used for stimulation between the two hemispheres at the bregma level, downwards until bone was reached, and the reading on the vernier scale noted.

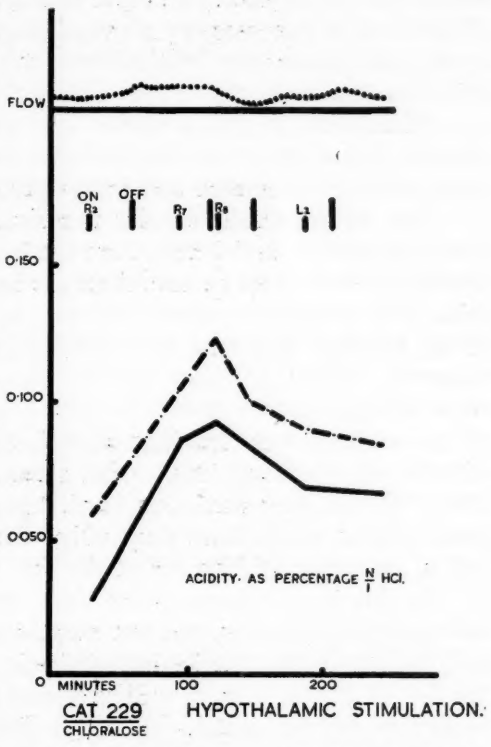

FIG. 647.—Hypothalamic stimulation. Stimulus: Thyratron at 50 per second. A graph showing acidity values in an animal in which the same procedure was followed as depicted in *Fig.* 646. On the right side the points stimulated in succession were: A, 2 mm. behind bregma, producing a sustained increase in flow and acidity. B, 12 mm. behind bregma, producing no appreciable effect; C, 14 mm. behind bregma, causing a diminution in flow and a fall in acidity.

Finally the left anterior region 2 mm. behind bregma was stimulated, causing an increase in flow and a check to the fall in acidity.

The stimulus used was by means of a thyratron valve discharging at a rate of 50 per second. The voltage used was just subminimal for the temporalis muscle, and the milliamperage in the region of 0·5. The whole of this rectangular block on each side of the midline was then 'sounded', and two sensitive spots were found. The first was situated 2 mm. behind the bregma, 2 mm. from the midline,

and 2 mm. from the base line. Stimulation at this point in every animal used for anterior stimulation almost invariably produced a response, and that response was always the same. A latent period, usually of about six minutes and sometimes as long as fifteen minutes, had to pass, and then a sustained increase in flow of gastric juice would occur, accompanied by an increase in acidity. Stimulation was usually carried on for some twenty to thirty minutes, and then, after cessation of the stimulus, this increased flow would persist for a variable time and then relapse again.

The physical properties of the juice so secreted resembled those of the secretion caused by transthoracic vagal stimulation, and if initially the flow from the resting stomach was mucoid, the anterior hypothalamic stimulation would change this to a much less viscous juice. In other words, the response obtained at this point was, to all intents and purposes, a pure vagal response. This effect could only be produced by stimulation in a very localized area with the strength of stimulus used throughout this series, and more than 4 mm. behind the bregma produced no response whatever.

Stimulation at this sensitive spot also brought about slowing of the heart, already demonstrated by Beattie, but in addition occasional syncope brought the experiment to an abrupt and premature close.

The second sensitive point was situated 14 mm. behind the bregma, 2 mm. from the midline, and 8 mm. from the base line. Occasionally, however, this point would be found to be 12 mm. from the bregma, but never any farther forward than this. The response obtained here was not as certain and as definite as stimulation at the anterior end, and in a number of experiments no response was obtained whatever. When a change was induced, however, that change was always of the same nature. In one or two animals the flow of juice, after a latent period of six to ten minutes, was definitely slowed, and this slowing was sustained as long as stimulation was proceeding. After cessation of the stimulus the flow would return slowly to the pre-stimulation level. Another phenomenon which was observed, and occurred much more frequently, was an increase in the production of mucus and a consequent falling off of the free acid content of the secretion.

In this hypothalamic series there were two cats which had had a complete transthoracic vagotomy, one ten months and the other six months previously. In both these animals anterior hypothalamic stimulation was successful in accelerating the flow of gastric juice, as well as raising the acidity, although not to such an extent as in the normal animal. Although the interval after vagotomy was long, post-mortem examination revealed no obvious regeneration, and one cannot but surmise that these vagal impulses were reaching the stomach via an alternative route. This physiological problem has not been followed up yet for confirmation.

In groups (*a*), (*b*), and (*c*) the experiments were repeated on the decerebrate animal in order to eliminate the possible effects of the chloralose anæsthetic, but in all the groups there was no difference in the response obtained. Decerebration was carried out in the usual manner under chloroform anæsthesia, all manipulations involved in the insertion of the collecting tube into the stomach being carried out before decerebration was performed.

COMMENTARY

The behaviour of the histamine response from the stomach is interesting in so far that the secretion produced is almost pure acid. Babkin (1930) and Vineberg (1935) both describe fully a detailed analysis of histamine juice, and come to the

conclusion that it must act on the parietal cell alone. The fact that the response is not materially affected by stimulation or section of the vegetative nerves, would confirm the belief that it acts directly on the cell itself, and not through any nerve-endings. Sugishima (1932) maintains that chloralose anæsthesia has a marked inhibitory action upon histamine, but this was not found to be the case in these experiments—no appreciable difference being noted between the response under chloralose or nembutal, or in the decerebrate cat.

The results obtained from the various procedures on the vegetative nerves to the stomach agree for the most part with those obtained by other workers. It has now been well established that nerve section does not permanently influence the normal resting acidity (Shapiro and Berg (1932), Ivy and Beazel (1936), Ferguson (1936), Madeleine Brown (1933), and others). In the same way, anatomists have long agreed that the peripheral autonomic nerves are mixed nerves, and the experiments described in this paper would strongly support such a contention. It was not found possible to repeat the experiments of Vineberg (1931), who maintains that stimulation of the vagus, using low frequencies in the region of 4 per second, produces mucus secretion. In the relatively few vagal stimulations which were carried out, it was found that no change in the gastric secretion could be obtained under about 25 stimuli per second, and at no time could mucus be induced to flow. The main reason for this apparently needless repetition of previous work was to obtain controls for the hypothalamic series under the same working conditions—a point which, in view of the conflicting results of others already referred to in the introductory paragraph, seemed advisable.

The findings in the hypothalamic area give a little more additional support to what is now a well-established physiological fact, that a higher centre for the autonomic nervous system lies in that region. The experimental work of Beattie, Brow, and Long (1930), Beattie (1932), Beattie and Sheehan (1934), Beattie and Kerr (1936), have established beyond doubt that the anterior region of the hypothalamus gives a parasympathetic response on stimulation, and the posterior hypothalamic area gives a sympathetic response.

The effects noted on gastric secretion are those which one would reasonably expect to happen—namely, that stimulation in the anterior part produces an increase in gastric secretion and a marked increase in acidity, with a tendency to a high proportion of free acid. The posterior region on stimulation produces a marked increase in the mucus content and a tendency to diminish the rate of flow and acidity. There seemed to be little tendency for acidity to rise as it did when the splanchnic nerves were stimulated, and it would seem probable that in the hypothalamus one is obtaining a more or less pure sympathetic response.

Between the two anterior and posterior sensitive regions the hypothalamus appears to be quite 'silent' as far as gastric secretion is concerned.

These two sensitive spots are situated in the region of the supra-optic and mamillary groups of nuclei respectively. Beattie has classified the hypothalamic nuclei into three main groups—viz., supra-optic, infundibular, and mamillary (*Fig.* 648). As far as one's experimental work is concerned, the hypothalamic region is too small, relative to the electrodes used, to be certain which individual nuclei of these groups are being stimulated. Even using the extremely weak current previously described, the histological changes are found to extend over an area of about 3 mm. spherical from the point of the electrode, and all that one is entitled to say is that stimulation

of the supra-optic group of nuclei gives a vagal response, and of the posterior group gives a sympathetic response, from the gastric mucosa.

The observations on the behaviour of mucus are extremely interesting, and casual references to it have previously been made by a number of workers (Toby, 1936; Webster, 1931). In experimental surgery one has always been led to believe that the secretion of mucus by a stomach was due: (1) To the fact that the general health of the animal was not all that it should be; (2) To prolonged starvation in some way; or (3) To the stomach becoming tired.

In the human stomach mucus is produced in quantity in chronic gastritis, whatever that all-embracing term implies, and is also noticed in extremely nervous patients who are undergoing test-meal investigations. This last observation seems to be quite important.

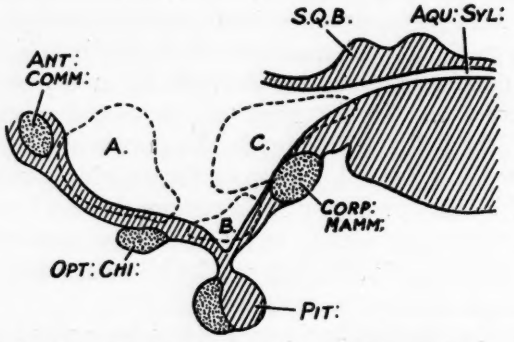

FIG. 648.—Schematic drawing of hypothalamic region: A, Supra-optic group; B, Infundibular group; C, Posterior or mamillary group. (*After Beattie.*)

The experimental work which has been done personally would suggest that there is some ground for believing that the formation and secretion of mucus is, in part at any rate, under nervous control. In all the acute series of experiments this type of secretion was induced with uncanny frequency by stimulation of the sympathetic part of the autonomic outflow.

It has been produced by splanchnic and cœliac ganglion stimulation within the abdomen, by splanchnic stimulation within the thorax, and finally by stimulation of the posterior hypothalamus, which part of the interbrain has now been investigated sufficiently by many workers to conclude that it contains a centre which, on stimulation, gives a purer sympathetic response than in any other part of the autonomic nervous system now known. The significance of this observation on mucus secretion, if it be confirmed and established, might afford a new approach to the old question of the trophic influences of the vegetative nerves of the stomach. The protective action of mucus is a widely accepted fact, and it would indeed be interesting if its formation were due to a vital function, and not merely due to local disturbances in the gastric mucosa.

CONCLUSIONS

1. The response of the gastric secretion to histamine is independent of nervous influences.

2. The secretion of mucus by the stomach is, in part at least, under the control of the sympathetic part of the autonomic nervous system.

3. Further evidence is adduced that the splanchnic is a mixed nerve.

4. Localized anterior and posterior hypothalamic stimulation produces parasympathetic and sympathetic effects on the gastric secretion.

My thanks are due to Sir Arthur Keith and Professor John Beattie for their kind help and criticism; and to Messrs. Watson and Steward for their technical assistance.

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THE NERVOUS FACTOR IN TRAUMATIC SHOCK*

By DAVID SLOME
McKENZIE McKINNON RESEARCH SCHOLAR
AND LAURENCE O'SHAUGHNESSY

INTRODUCTION

THE experimental observations recorded in our previous papers were undertaken to define the causal agencies involved in the circulatory collapse following trauma. Trauma was inflicted by blows to the one hind limb of the anæsthetized experimental animal, producing fracture of the femur, together with damage to the muscles of the thigh. We adopted this method of trauma as it paralleled the following typical case observed in practice: A 6-year old boy was run over by a bus—the vehicle passed over both thighs—and he was admitted to hospital in a state of shock. In this state he died some twenty-four hours later. At autopsy there was much extravasation of blood into the muscles around the fractured ends

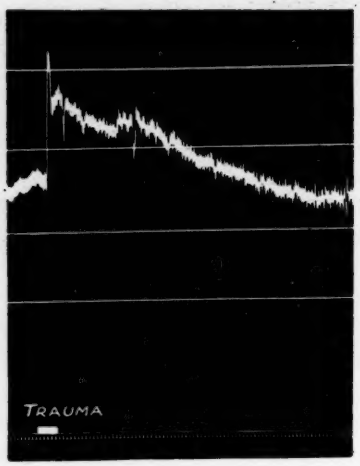

FIG. 649.—Initial pressor effect of trauma.

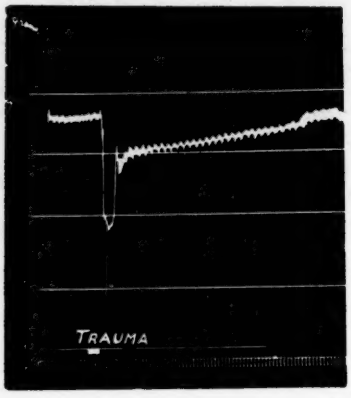

FIG. 650.—Initial depressor effect of trauma.

of the femora, and this extravasation extended into the retroperitoneal tissues. There was intense pallor of the skin. The omentum, intestines, liver, and kidneys were also pale in colour, and on section of these organs the cut surfaces did not bleed. There was no evidence of congestion of the splanchnic vessels, nor were any petechial hæmorrhages observed. The lungs were pale, and the heart was small, contracted, and only moderately full of blood.

The immediate effect of trauma to one hind limb on the blood-pressure of

* This work was carried out at the Buckston Browne Research Farm and the Bernhard Baron Research Laboratories of the Royal College of Surgeons of England, under the direction of Professor J. Beattie. One of us (L. O'S.) is in receipt of a personal grant from the Medical Research Council.

the experimental animal is either a pressor or a depressor response. When the initial effect is pressor the blood-pressure returns to its pre-traumatic level within a few minutes of the cessation of trauma. This is well shown in *Fig.* 649. When the primary effect is depressor, the blood-pressure is again usually restored fairly rapidly to its previous level or just below that level (*Fig.* 650). Whether the initial effect is pressor or depressor, a gradual decline of the blood-pressure sets in, generally about half to one hour after trauma. The blood-pressure now declines steadily till the death of the animal occurs, within three to four hours after trauma (*Fig.* 651). When once this secondary progressive fall of blood-pressure has set in and reduced the pressure to a shock level, recovery has never been observed in any experiment in our series.

This analysis of the effect on blood-pressure of trauma into initial and late effects is not intended to support the differentiation of clinical traumatic shock into primary and secondary, which has, in our view, merely served to confuse the consideration of the problem.

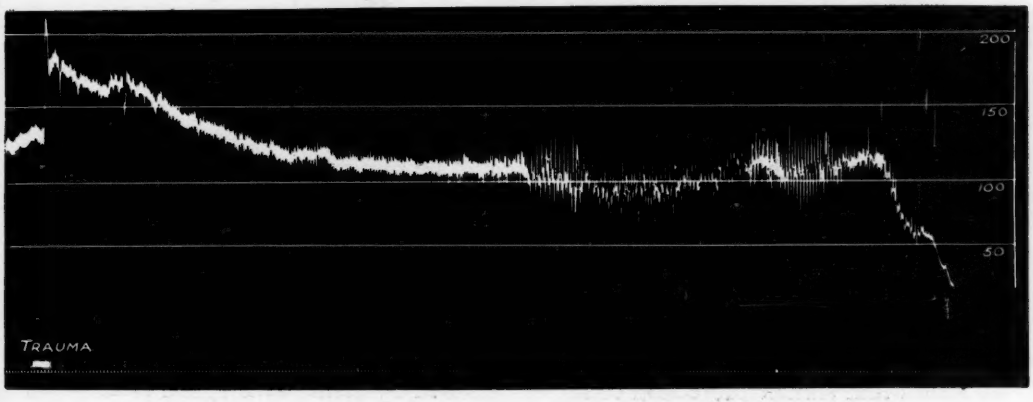

FIG 651.—Showing secondary steady decline in blood-pressure up to the time of death.

We demonstrated in our first series of experiments[3] that :—

1. The amount of depressor substances which can be extracted from the tissue subjected to trauma was insufficient to produce the phenomena of shock.

2. Complete occlusion of the venous return from a limb, thus preventing the absorption into the circulation of depressor toxins, does not prevent the development of shock.

3. Perfusion of a traumatized limb failed to produce any evidence of the presence in the traumatized region of vasodilator substances.

4. Experiments designed to demonstrate the presence of depressor toxins in the systemic circulation of the shocked animal were unsuccessful.

5. The post-mortem appearances in animals dead from shock differs strikingly from those seen after death from histamine poisoning.

We therefore concluded that the theory of traumatic toxæmia could not be substantiated.

More recently Roome and Wilson[4] have shown that the depressor effects of injection of extracts of traumatized tissue recorded by some investigators as evidence for the toxic theory, are due to blood coagulation and to particles of fat and tissue

in these extracts; when the extracts were centrifuged before injection, no depressor effect was observed.

Dragstedt and Mead[2] obtained constantly negative results in attempts to demonstrate any histamine-like substance in the blood and thoracic duct lymph in animals in a state of traumatic shock. These results are most striking when it is recalled that, employing the same technique, they were able to show the presence of such a substance in the blood in anaphylactic shock. As a further control they were able to show that if histamine is infused intravenously in sufficient amount to cause shock, its presence in the circulating blood was demonstrable by their methods.

Toxæmia having been eliminated as a causal factor, we were left with the two remaining factors—fluid loss at the site of trauma, and the production of nervous impulses in that area.

In our early series of experiments we measured the fluid loss by the method of Blalock,[1] and found that it was not sufficient to permit the conclusion that the fluid loss was the sole factor determining the decline of the blood-pressure. This conclusion has been confirmed in a further series of control experiments.

There is also no direct relation between the survival time of the animal and the amount of the fluid loss. Animals which have suffered a relatively large fluid loss may survive for a considerable time; others may die very rapidly with a very small fluid loss.

It was known that the onset of shock could be prevented by complete occlusion of the arterial supply of the limb prior to trauma. In this case fluid loss cannot occur; we showed that in the presence of such a complete 'ischæmia', the limb is also 'anæsthetic' and the nervous factor cannot operate. When, however, this 'ischæmic' limb was supplied with blood by cross circulation from a second animal trauma produced shock. We confirmed the work of Simonart[5], who found that section of the nerves of the limb before trauma prevented the onset of shock, and we found that similar protection could be obtained by continuous spinal anæsthesia.

When trauma is applied to the hind limb of an animal kept under continuous spinal anæsthesia, an initial depressor effect may be observed at the moment of trauma (if fluid loss is marked), but the blood-pressure is soon restored and the secondary decline to shock level does *not* occur. The blood-pressure remains well above a shock level as long as the anæsthesia is maintained.

It was concluded that in the type of traumatic shock investigated, the local production of a toxin plays no part in the syndrome. Traumatic shock is caused by the operation of two factors: fluid loss into the traumatized area; and nervous impulses from that area.

In the series of 103 experiments reported in this paper the toxic theory is not considered, and our attention has been confined to establishing the role and relative importance of the fluid loss and the nervous factor.

THE EXPERIMENTS

1. Cross-circulation Experiments.—In our first series of experiments we tested the effects on the blood-pressure of trauma to the hind limb of one animal (A) when that limb is being supplied with blood from a second animal (B) and completely excluded from its own circulation (*Fig.* 652). Any effect on the

blood-pressure of animal A can only be attributed to the effect of nervous impulses. In these cross-circulation experiments trauma produced a fall of blood-pressure to shock level and death of animal A, although only nervous pathways connect the traumatized limb with the rest of that animal.

We have repeated this type of experiment and confirmed this result (*Fig.* 653).

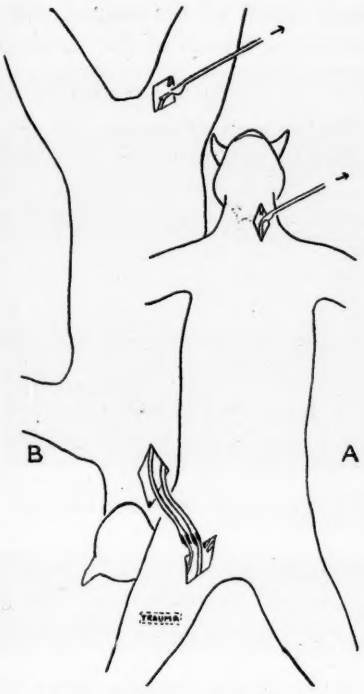

FIG. 652.—Diagram of cross-circulation experiment (*see* text).

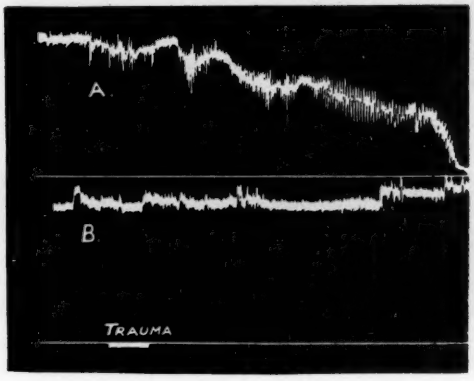

FIG. 653.—Blood-pressure tracings of cross-circulation experiment. Signal: Trauma to limb of animal A supplied with blood by animal B. Gradual decline of blood-pressure of A to death, B only slightly affected.

2. Perfusion Experiments.—The difficulties associated with establishing such a cross circulation, however, prompted us to use a simpler method of keeping the tissues of a traumatized limb alive by perfusion with an oxygenated hæmoglobin-Ringer solution (Amberson-Höber), using a Dale-Schuster pump.

In these experiments, the skin of the hind limb of the anæsthetized cat was separated from the skin over the lower abdomen and back by a circular incision with careful hæmostasis. The vascular bed of the limb was disconnected from the rest of the animal by ligating the femoral vessels at Poupart's ligament and by a series of interlocking stout ligatures round the upper ends of all the muscles and tissues of the thigh. The nerves previously isolated were excluded from these ligatures. The femoral artery and vein were cannulated and connected to the Dale-Schuster pump, which supplied the limb with oxygenated hæmoglobin-Ringer solution. In all these experiments the practically complete discontinuity of the circulation of the limb and the rest of the animal was established at post mortem. Direct electrical faradization of the fine digital nerves of the toes and of the muscles of the limb served to demonstrate that the tissues of the perfused limb were receptive and capable of reacting to stimulation. The effect of trauma to such a limb on the blood-pressure of the animal recorded in the carotid artery has been investigated.

In one group of experiments the nerves of the limb were preserved intact, while in another group the nerves were sectioned before the limb was traumatized.

Figs. 654–656 are typical blood-pressure tracings of these two types of experiment. Trauma to a limb thus kept alive and reactive by perfusion, with the nervous connections between the limb and the rest of the animal intact, produces a gradual decline of the blood-pressure and ultimately death of the animal after about four to five hours (*Figs.* 654, 655). The appearances typical of the shocked state were seen at post-mortem examination in these experiments.

FIG. 654.—Effect on blood-pressure of animal of trauma to limb kept alive by perfusion. Nerves of limb intact. Initial blood-pressure 160 mm. Hg. Death 4½ hours after trauma.

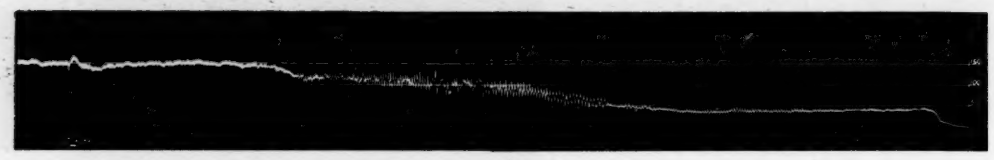

FIG. 655.—Effect on blood-pressure of animal of trauma to limb kept alive by perfusion. Nerves of limb intact. Initial blood-pressure 150 mm. Hg. Death 4 hours after trauma.

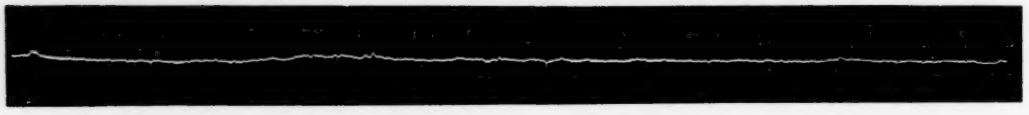

FIG. 656.—Effect on blood-pressure of animal of trauma to limb kept alive by perfusion. Nerves of limb sectioned. Initial blood-pressure, 130 mm. Hg. Final blood-pressure 100 mm. Hg. 5 hours after trauma.

When the nerves of the perfused limb are cocainized or severed before trauma, the blood-pressure remains well above a shock level, and, in the experiment illustrated in *Fig.* 656, the blood-pressure before trauma was 120 mm. Hg and was still at 100 mm. Hg five hours after trauma. At autopsy, after the termination of this experiment, the viscera and omentum were normal.

BLOOD TRANSFUSION IN EXPERIMENTAL TRAUMATIC SHOCK

Further evidence for the operation of nervous factors in traumatic shock is provided by the effects of blood transfusion in the state of shock experimentally produced.

When the blood-pressure has declined to a shock level, transfusion of blood in an amount approximately equal to the actual fluid loss into the traumatized limb will not permanently restore the blood-pressure. This is well illustrated in the experiment shown in *Fig.* 657. In this experiment the initial blood-pressure was 150 mm. Hg. After trauma the blood-pressure declined to 75 mm. Hg. At this point 50 c.c. of blood were transfused intravenously. The blood-pressure rose rapidly to its pre-traumatic level, but soon declined again, and after half an hour had fallen to 80 mm. Hg. The fluid loss at the termination of the experiment was only 56 c.c.

The inefficacy of blood transfusion in this experiment cannot be attributed to the blood supplied leaving the circulation by leaking into the traumatized tissues, as the fluid loss at the close of the experiment was no greater than the amount of blood transfused.

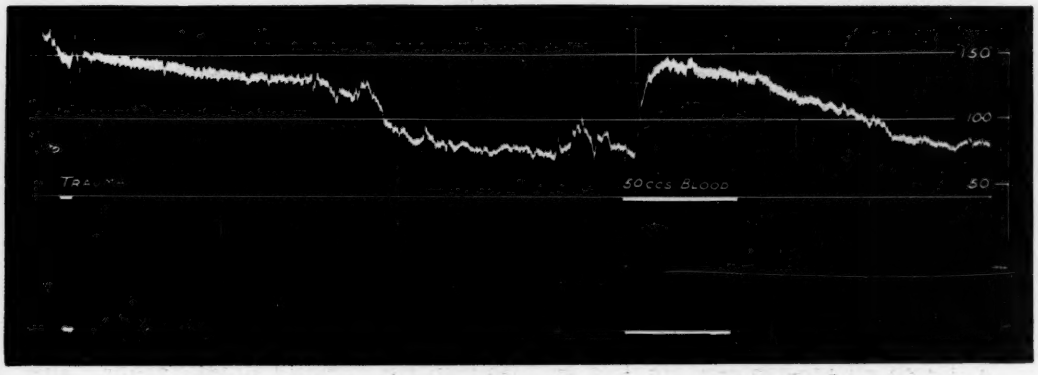

FIG. 657.—Trauma: Blood transfusion. Initial blood-pressure 150 mm. Hg. Signal 1: Trauma. Gradual decline of blood-pressure. Signal 2: Blood-pressure 75 mm. Hg. Blood transfusion, 50 c.c. After 30 minutes blood-pressure again fallen to 80 mm. Hg.

BLOOD TRANSFUSION AND SPINAL ANÆSTHESIA IN TRAUMATIC SHOCK

In a further series of experiments we were able to show that if the transfusion of blood was accompanied by spinal anæsthesia, the rise of blood-pressure produced by the transfusion was maintained. The blood-pressure tracing of an experiment of this type is shown in *Fig.* 658.

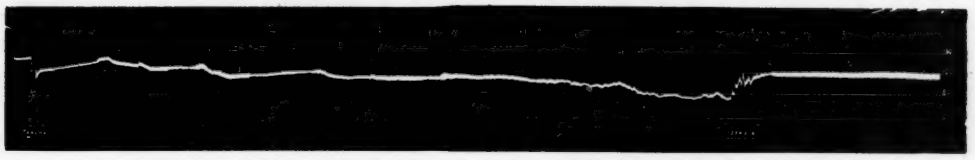

FIG. 658.—Trauma: Blood transfusion: Spinal anæsthesia. Initial blood-pressure, 130 mm. Hg. Signal: Trauma. Decline of blood-pressure to 40 mm. Hg. Signal 1–5: Blood transfusion, 50 c.c. Signal 6: Spinal anæsthesia. Blood-pressure rises to 100 mm. Hg and is maintained at this level.

In this experiment the initial blood-pressure was 130 mm. Hg. Two hours after trauma the blood-pressure was 40 mm. Hg. Fifty c.c. of blood were transfused intravenously. Immediately after the transfusion spinal anæsthesia was induced. The blood-pressure rose as a result of the transfusion to 100 mm. Hg, and remained steady at this level.

AMPUTATION OF TRAUMATIZED LIMB OF SHOCKED ANIMAL

The effect on the blood-pressure of the shocked animal of amputation of the traumatized limb was tested. The results of these experiments are seen in *Table I.*

Table I.—SHOWING EFFECT OF AMPUTATION OF TRAUMATIZED LIMB ON BLOOD-PRESSURE OF SHOCKED ANIMAL

No. of Cat	Weight	Blood-pressure at Time of Trauma	Amputation			Result
			Time since Trauma	Blood-pressure	Anæsthetic	
1	3250 g.	130 mm. Hg	1½ hr.	60 mm. Hg.	Spinal	Killed 2 hr. after amputation. Blood-pressure 130 mm. Hg. Fluid loss, 47 c.c.
2	3000 g.	130 mm. Hg.	1½ hr.	70 mm. Hg.	Spinal	Killed 2½ hr. after amputation. Blood-pressure 120 mm. Hg. Fluid loss, 50 c.c.
3	2500 g.	110 mm. Hg.	1¼ hr.	55 mm. Hg.	—	Died 1½ hr. after amputation
4	4000 g.	120–130 mm. Hg	2 hr.	60 mm. Hg.	Spinal	Killed 2 hr. after amputation. Blood-pressure 100/110 mm. Hg. Fluid loss, 66 c.c.
5	3000 g.	150 mm. Hg	1½ hr.	70 mm. Hg.	—	Killed 3 hr. after amputation. Blood-pressure 110 mm. Hg

In the majority of these experiments amputation is followed by a marked improvement of the animal's condition. The beneficial effect of the amputation may be due to the cessation of fluid loss or to the removal of the source of origin of the afferent depressor nervous impulses.

We have measured the fluid loss at varying intervals after trauma, and find that the bulk of the blood-loss occurs immediately after trauma. This may account for the sudden precipitous fall of pressure which may occur during and immediately after trauma. From this fall the blood-pressure can be restored by the compensatory mechanisms of the body. When the blood-pressure has fallen to shock level the fluid loss has nearly all occurred, so that the beneficial effect of experimental amputation cannot readily be attributed to the prevention of further fluid loss. It is much more probably due to the removal of the source of the barrage of nervous impulses from the injured area.

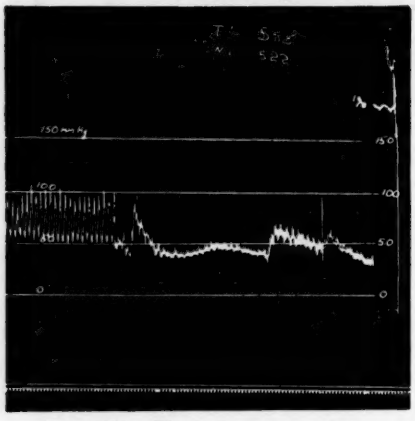

FIG. 659.—Blood-pressure tracing two hours after trauma. Oscillations in blood-pressure level. Signal: Section of nerves of traumatized limb. Oscillations disappear.

In our last paper we postulated the existence of a continuous barrage of nervous impulses arising in the traumatized area, and we suggested that the large variations of the systemic blood-pressure level of the shocked animal might be attributed to the operation of these impulses since the

variations are abolished by spinal anæsthesia. In support of this view we have been able to show that these fluctuations in the level of the blood-pressure disappear when the nerves of the traumatized limb are cut (*Fig.* 659).

To obtain more direct evidence for the existence of the barrage of nervous impulses we have taken records of the action currents in the nerves of a traumatized hind limb, and compared these with similar records from the other untraumatized limb. Records were taken before trauma, five minutes after trauma, and then at regular intervals after trauma till the death of the animal. We were unable to obtain a record during the actual trauma. Comparison of these records after trauma with the resting discharge in the same nerve before traumatizing the limb, shows that no abnormal impulses are recognizable for three-quarters of an hour to one hour after trauma. After this time an almost continuous barrage of impulses

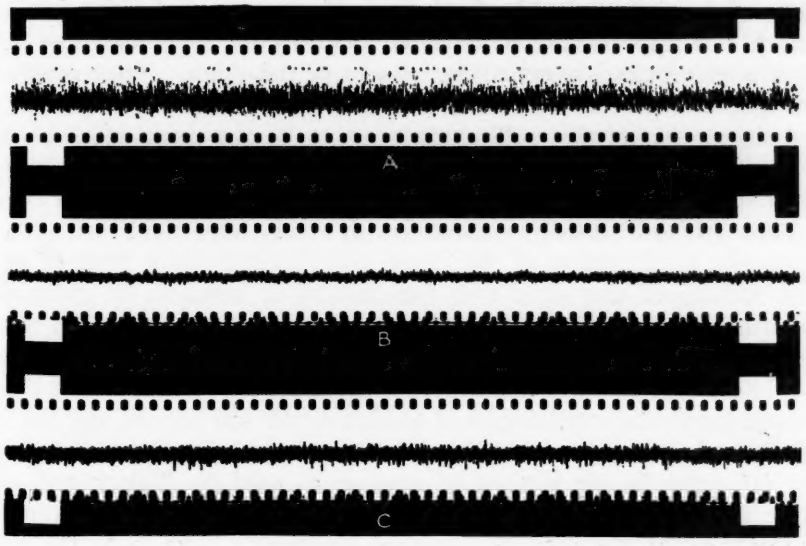

FIG. 660.—Records of nerve impulses. A, Traumatized limb two hours after trauma. B, Disappearance of impulses after section of nerve peripheral to electrodes. C, Record for same nerve of untraumatized limb.

is rapidly developed (*Fig.* 660, A). These can be shown to be centripetal afferent impulses by ligaturing or sectioning or novocainizing the nerves peripheral to the electrodes used for picking up the nerve action potentials (*Fig.* 660, B).

The absence of any impulses for a period after trauma, and their appearance after this time and persistence till the death of the animal, accords accurately with the gradual decline of the blood-pressure which sets in at this time. These records afford striking confirmation of our experimental investigations, which have shown that this fall must be due to the activity of nervous influences.

The detailed description of these records forms the subject of a separate communication.

DISCUSSION

It is perhaps well to emphasize at the outset that we are solely concerned with the cause of death in the experimental animal subjected to the type of trauma

described in the introduction. Our experiments are directed to establishing the nature of the link between the traumatized area and the rest of the animal. Such observations as we have made on other metabolic changes in the state of shock are not dealt with in this paper. While they may prove of value in the final interpretation of the nature of the syndrome, they are obviously secondary to the operation of the primary factor.

There is now almost general agreement that the theory of traumatic toxæmia can be abandoned, and the duration of our experiments and the manner in which they were performed did not allow of the onset of the bacterial toxæmia which, as a natural result of gas gangrene, played so important a part in supporting the theory of traumatic toxæmia when this was revived during the war. An animal will very readily and fairly rapidly restore its blood-pressure after a loss by simple hæmorrhage of one-quarter of its blood volume, and yet in many of our experiments a fluid loss of one-sixth of the blood volume has been associated with a fatal outcome.

Blalock and his co-workers[1] measured carefully the local fluid loss at the site of trauma after repeated blows to the *muscles* of the thigh. They concluded that the local fluid loss due to extravasation of blood and exudation of plasma into the lacerated tissues was always sufficiently great to be the cause of shock following injury.

While severe trauma to very large masses of muscle as reproduced experimentally by Blalock is undoubtedly associated with a very large fluid loss, such injuries constitute only a proportion of the cases seen clinically in a state of shock. It cannot be maintained that all or even the majority of instances of traumatic shock are accompanied by a sufficiently large fluid loss to cause death. In some cases profound shock is associated with very trivial injuries, with very little damage to muscle. In the type of case which is frequently seen, the severity of the shock is out of all proportion to the small degree of trauma to the soft tissues, and the fluid loss is obviously inadequate to account for more than a very minor part of the condition.

The difference in the method of trauma, as used by Blalock and ourselves, probably explains the fact that in our experiments, both previously reported and in the present series, the fluid loss is never sufficiently large to be the sole factor accounting for the death of the animal, as claimed by Blalock. Even in the experiments in which amputation or control of the nervous factor has resulted in recovery, the restoration of the blood-pressure has always been slow. This discrepancy must be regarded as a result of the operation of the barrage of nervous impulses, whose existence we have established. These impulses arise from the traumatized area, not at the moment of trauma, but later in the post-traumatic period. We have not as yet investigated the manner in which the barrage of nervous impulses operates, but direct indication of its existence has been provided by oscillographic records. Evidence of the importance and lethal effect of the barrage is provided by the perfusion experiments, and the beneficial effect of its removal is demonstrated by the amputation experiments.

The initial effect of trauma is either a pressor effect or a depressor effect. The latter effect is observed when there is definite marked muscle damage with consequent large fluid loss. This initial fall is absent: (1) In the case of trauma with arteries to the limb ligatured; and (2) In trauma to a limb kept alive by cross-circulation experiments or by perfusion with the Dale-Schuster pump. This initial fall is present in experiments where the nervous factor is controlled. It is present

when trauma is applied (1) after spinal anæsthesia, or (2) after cocainization or section of nerves from the limbs traumatized. The initial fall in pressure, when present, must be attributed therefore to the loss of fluid from the circulation and to hæmorrhage into traumatized tissues.

The subsequent gradual decline of the pressure cannot be attributed to the continued operation of the fluid loss, as it is absent (1) when trauma is applied under spinal anæsthesia, (2) when trauma is applied to denervated limb, and (3) when perfused denervated limb is traumatized; and it is present when the nervous factor is operating, but when no fluid loss can occur, as in the experiments when trauma is applied to a limb kept alive by cross circulation or perfusion and connected to the animal only by neural connections. The initial depressor effect of trauma is due to fluid loss; the significant secondary decline to shock is caused by the continued and continuous discharge of nervous impulses from the traumatized area (*Fig.* 661). The nervous factor operating alone can cause death. The fluid loss in these experiments, provided the nervous factor is controlled, is not fatal. We regard this concept of the late development and persistence of the nervous barrage as of fundamental importance in our understanding of the aetiology of surgical shock.

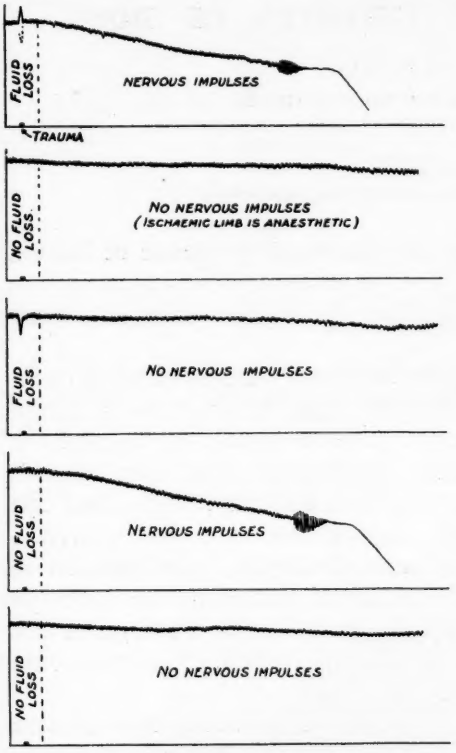

FIG. 661.—Diagram of effect on blood-pressure of: (1) Trauma to hind limb; (2) Trauma after occlusion of blood-supply; (3) Trauma to limb with continuous spinal anæsthesia; (4) Trauma to perfused limb; (5) Trauma to perfused denervated limb.

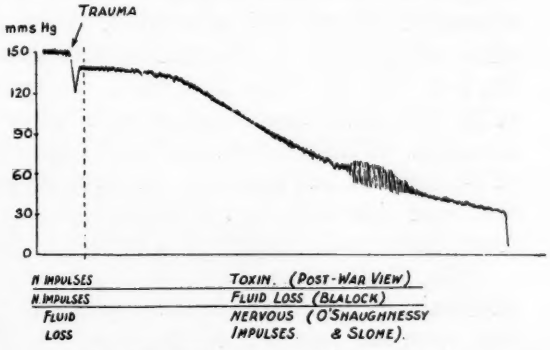

FIG. 662.—Diagram summarizing the views on the aetiology of shock.

Grateful acknowledgement is made to Dr. J. Trevan and Dr. A. C. White of the Wellcome Physiological Research Laboratories for a supply of hæmoglobin solution.

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SHORT NOTES OF RARE OR OBSCURE CASES

MULTIPLE DISSIMILAR NEW GROWTHS OF BONE

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A CASE of the simultaneous occurrence of several dissimilar neoplasms of bone is sufficiently rare to merit record.

P. S., male, aged 48, a cultivator, was admitted into the Medical College Hospitals under the care of one of us (P. N. R.).

HISTORY.—About the latter part of 1933 he noticed a painless swelling of his right thigh above the knee. About the middle of 1934 he sustained a simple fracture of the femur at this site after a trivial accident which he described as a stumble on the bank of a pond. Firm union occurred in about three months, after which period he was able to walk about with some difficulty. Since this date the limb was never free from pain. In January, 1935, he noticed a painful swelling of his left collar-bone, which he ascribed to his habit of carrying heavy loads on his shoulder. Within a few months he began to complain of pain along the inner side of his left arm and forearm. In February, 1936, he noticed some weakness in gait, and soon afterwards loss of control over his bladder and bowels. Paralysis of the lower limbs was complete in a month or two.

ON ADMISSION.—The patient presented a cachectic appearance, with bilateral proptosis, dyspnœa and cyanosis, marked enlargement of the vessels of the neck, and complete paralysis of the lower extremities. Temperature, 101° F.; pulse-rate, 126/44.

PHYSICAL EXAMINATION.—

Right Femur: The lower third of the femur appeared moderately expanded and pyriform in shape. The longitudinal and transverse diameters were nearly $6\frac{1}{2} \times 4\frac{1}{2}$ in. respectively. There was an old malunited fracture with shortening of $1\frac{1}{2}$ in., and typical eversion of the foot. There were numerous and prominent veins in front of the knee and the lower third of the thigh. There was also an aneurysm of the popliteal artery. The knee-joint was free, and could be bent to a little over 90°.

Left Clavicle: The neoplasm occupied the inner three-fourths of the clavicle, the supraclavicular fossa, and the first intercostal space. There were numerous large veins over the tumour, and it was 5×4 in. in dimension.

Paraplegia: The level of paresis extended up to the 5th intercostal space. Anæsthesia and paralysis were complete below the level of the umbilicus. *Bladder*: Retention with overflow. *Rectum*: Involuntary motions.

Heart: Apex beat in the 6th left interspace, ¾ in. outside the nipple line. There was a systolic bruit in the mitral area. The left radial pulse could be barely palpated.

Lungs: Crepitations were present over both the bases.

LABORATORY INVESTIGATION.—

Hæmatological Examination.—

Hæmoglobin	45 per cent
White blood-corpuscles	11,700 per c.mm.
Polymorphonuclears	80 per cent
Monocytes	2 per cent
Lymphocytes	15 per cent
Eosinophils	0 per cent
Wassermann reaction	Negative

Urine Analysis.—

Albumoses	Present
Bence-Jones albumoses	Nil

Blood-pressure.—

Right arm	110/55 mm. Hg
Left arm	90/50 mm. Hg

Blood-chemistry.—

Calcium: 11 mg. per 100 c.c.
Phosphorus: could not be determined.

X-RAY EXAMINATION.—Owing to the extremely weak state of the patient, it was neither possible nor advisable to conduct as thorough an examination radiologically as one would have liked. The series of radiographs show the following:—

Skull: (1) A heart-shaped area of bone absorption, roughly centrally placed in the frontal side of the coronal suture and $1\frac{1}{2} \times 1\frac{1}{2}$ in. in size.

FIG. 663.—Radiograph of skull, showing areas of bone absorption.

(2) Immediately to the right of this, another similar area, about $\frac{3}{4} \times \frac{3}{4}$ in. in size. (3) A third similar area in the right parietal bone, 1 in. behind the coronal suture, near the sagittal plane. These areas are all suggestive of metastasis from a neoplasm (*Fig.* 663).

Left Clavicle: The whole of this bone, excepting about an inch of the outer end, has been destroyed. A rounded opacity, roughly 5 in. in diameter, is seen about it. This is occupied by an irregularly ossified mass in the centre, with radiating spicules, characteristic of osteogenic sarcoma. (*Fig.* 664.)

Ribs: The first rib on the left side is involved in the clavicular lesion, and presents considerable destructive changes. The outer part of the bony portion of the third rib shows evidences of cavitation and some bone absorption with bony reaction.

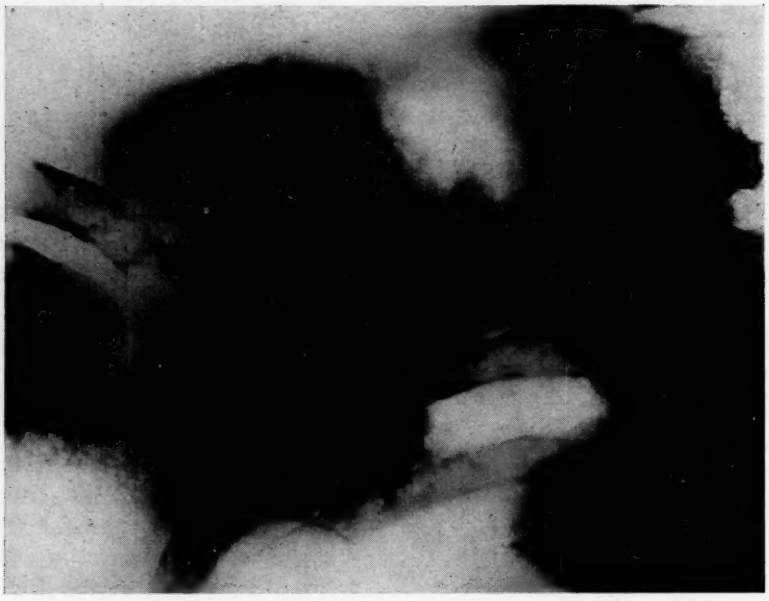


FIG. 664.—Radiograph of shoulder, showing almost total destruction of the clavicle. Note the rounded opacity, occupied by an ossified mass with radiating spicules, characteristic of osteogenic sarcoma.

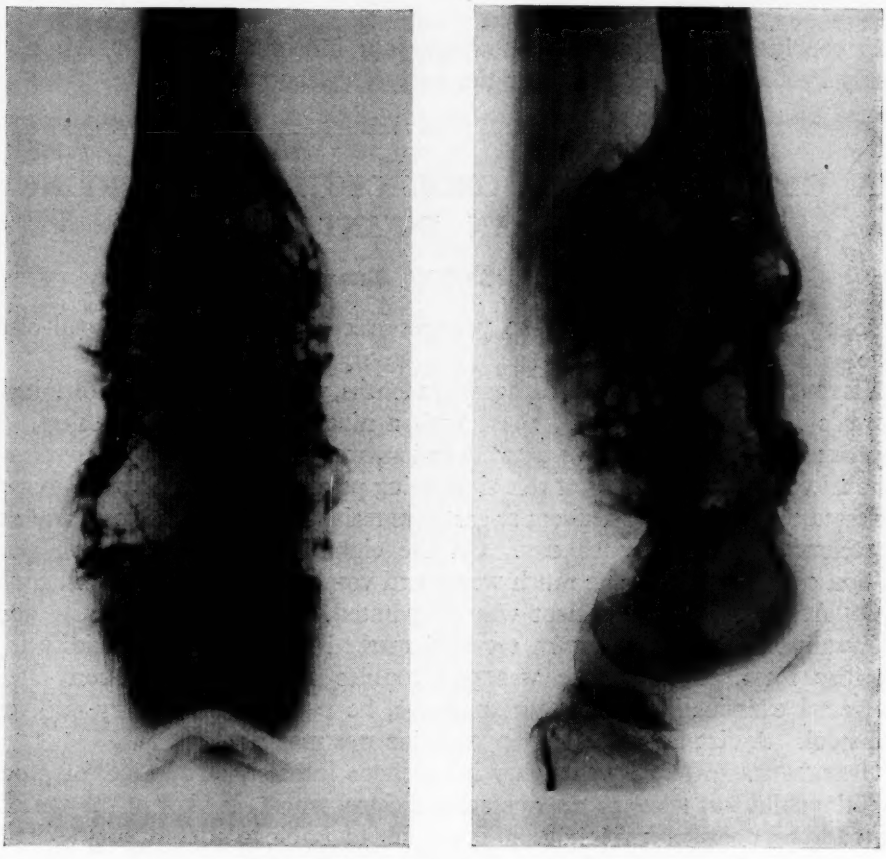
Scapula: The upper border is not seen; it appears to be involved in the clavicular lesion. An area of bone destruction with clear-cut borders, about $\frac{3}{4}$ in. in diameter, is seen near the inferior angle of the scapula. Here, as in the cranial lesions, there is no evidence of any bony reaction.

Right Femur: At the lower third of the diaphysial portion of the bone are seen the following: (1) Expansion of the bone, with coarse trabeculation and cavitation. (2) The outer bony shell has been completely destroyed. (3) Evidences are present of an old and united fracture—apparently pathological. (4) At the periphery of this irregular expanded mass is seen marked spiculation of bone, particularly on its posterior aspect. (*Figs.* 665, 666.)

Spine: Irregular flattening of the vertebral bodies, with areas of bone absorption, is seen in the 4th, 5th, 7th, 8th, 9th, and 12th dorsal vertebræ. Except in the case of the 4th and 5th dorsal vertebræ, the intervertebral discs do not appear

involved and show no diminution in spacing. No evidence of any bony reaction is seen in the dorsal spine, except faintly in the case of the 12th dorsal body.

The first lumbar vertebra is much flattened. The second and the third lumbar vertebræ do not show this flattening, but the bodies are irregularly cavitated and in places show irregular sclerosis of bone. Trabeculation is marked in these two vertebral bodies. The 4th vertebral body has preserved its outline, but it shows evidences of rarefaction generally and some bone reaction, somewhat spiculated on



FIGS. 665, 666.—Radiographs of right femur, showing expansion of bone, with coarse trabeculation and cavitation, and an old fracture.

the left side of its upper surface. The fifth lumbar vertebra is flattened and rarefied, and the bony architecture is largely destroyed.

The transverse processes of the third lumbar vertebra are both irregularly expanded and marked with signs of cavitation and trabeculation, the latter particularly on the left side. The bone is also rarefied to some extent.

Chest and Lungs: No metastatic appearances.

RADIOLOGICAL DIAGNOSIS.—

The appearances are suggestive of the following:—

1. An osteoclastoma of the lower third of the right femur, with a malunited pathological fracture. The neoplasm appears to have undergone changes at its periphery, which now strongly resemble those seen in an osteogenic sarcoma.

2. An osteogenic sarcoma of the left clavicle which has invaded and destroyed the left first rib and the upper border of the left scapula.

3. Metastatic deposits with destruction of bone in: (*a*) The 3rd left rib; (*b*) The inferior angle of the scapula; (*c*) The skull; and (*d*) The dorsal and lumbar vertebræ. The metastases in the latter are of extreme interest, as they appear to be of two types: in the 2nd and 3rd lumbar vertebræ the changes seen resemble those of osteoclastoma, whereas in the others a more destructive type of lesion is noticed, as in osteogenic sarcoma.

In conclusion, we wish to acknowledge our indebtedness to Dr. M. N. De, formerly Professor of Pathology, for the necessary laboratory data.

A CASE OF ACUTE CHOLECYSTITIS OCCURRING IN AN INFANT

By V. J. DOWNIE, Birmingham

The following is believed to be the youngest recorded case of acute gall-bladder disease.

History.—T. W., a male child, aged 15 months, was admitted to the Birmingham Children's Hospital on Sept. 9, 1937, with a nine days' history of illness. The first phase of the illness, of five days' duration, was characterized by diarrhœa and occasional vomiting, the stools at this time being pale and loose. This was regarded and treated by his medical attendant as gastro-enteritis, and considerable improvement had occurred by the seventh day. On the eighth and ninth days the general condition of the child became much worse and vomiting was frequent and copious.

On Admission.—The patient was dehydrated, listless, and obviously acutely ill. Examination of the abdomen revealed marked tenderness and rigidity in the right iliac fossa. Temperature was 99·6°, pulse 130, and respiration rate 24. After rectal examination, which was negative, he passed a small very dark green viscid stool. A diagnosis of acute appendicitis was made.

Operation.—After a preliminary intravenous infusion of 200 c.c. of glucose-saline, the child was taken to the operating theatre, where, in view of the age of the patient and his poor general condition, it was felt advisable to attempt simple drainage of the supposed appendix abscess through a small gridiron incision under local anæsthesia. However, on opening the abdomen there was a gush of clear free fluid and a substantially normal appendix was readily displayed. The visceral peritoneum was somewhat congested, and a finger in the abdomen encountered a smooth mobile mass extending down to well below the umbilicus; it was described as feeling "like a gall-bladder or an intussusception".

Further exploration was obviously necessary, and under ether anæsthesia a right paramedian incision was hurriedly made. The mass presented, and was seen to be a greatly distended gall-bladder, the wall of which was œdematous and of a mottled black, brown, and green appearance. Thirty c.c. of nearly black bile were aspirated and a No. 8 catheter was sewn into the gall-bladder, when a further three ounces of the same viscid bile drained on the table. There was appreciable thickening of the wall of the organ, but no apparent lymph exudation or palpable

adhesions. In view of the patient's condition, no further exploration of the anatomy of the part was attempted, and after introducing a tube into the pelvis via the gridiron incision both wounds were closed.

SUBSEQUENT PROGRESS.—The following day no drainage occurred from the catheter and the diagnosis of torsion of the gall-bladder was considered, but on the second day normal golden bile began to discharge and became gradually paler during the course of the next few days; approximately 30 c.c. drained in the twenty-four hours. On the ninth post-operative day the catheter was removed, and the biliary fistula closed in three days. Both wounds healed by first intention, and convalescence was quite uneventful, except for some abdominal distension on the second and third days. Indeed, there seemed to be dramatic improvement in the general condition of the child following relief of the tension in the gall-bladder.

Cultures were taken from the bile on three occasions during convalescence, but in spite of care in collection of the material, the nature and variety of the organisms grown—namely, *Sta. albus* from the first swab, *B. coli* from the second, and *Sta. aureus* plus *B. coli* from the third—would suggest that all of these were possibly contaminants, and it cannot be stated with any certainty that one or other organism was the causative agent.

Comment.—It is felt that the cholecystitis in this case was of two days' duration and was a sequel to a preceding gastro-enteritis, and that the fundus of the gall-bladder at the time of operation was in the pre-gangrenous state, termed by Lobingien "necrotic œdema", the mottled appearance of the wall being caused by destructive changes in the cells resultant on blockage of the minute capillary and lymphatic drainage by œdema. The dark green coloration of the stools would seem to be due to the presence of altered bile, and the absence of drainage on the first post-operative day to œdema of the cystic duct or neck of the gall-bladder. The probable sterility of the bile is, curiously enough, a not uncommon finding in gangrene of the gall-bladder. In a series of cases collected by S. A. Vest, of the Surgical Department of the Johns Hopkins Hospital, the gall-bladder and contents were cultured in five cases of cholecystectomy for gangrene of the organ, the culture being sterile in each instance. It is interesting to note that two cases in this series were young boys, one aged ten and the other four.

I am indebted to Professor Bertram Lloyd, at whose direction I operated on this case, for permission to publish the notes.

INTERNAL HERNIA FOLLOWING POSTERIOR GASTRO-JEJUNOSTOMY

BY D. J. HARRIES, CARDIFF

SEVERAL cases of hernia of the small intestine through the opening made in the transverse mesocolon in a posterior gastro-jejunostomy have been recorded. In the greater number the hernia has been discovered months or years after the operation, when the patient has been re-admitted for intestinal obstruction resulting from this complication.

In this case the condition was accidentally discovered during an operation primarily carried out for resuture of the abdominal wall, which started giving way on the fourteenth day.

HISTORY.—F. B., a male aged 38, a civil servant, was admitted to the Cardiff Royal Infirmary on January 10, 1936, with a typical history of a chronic duodenal ulcer.

ON EXAMINATION.—He had some rigidity of the right rectus and slight tenderness to palpation in the right epigastric region. The X-ray examination was negative, but the patient was an intelligent individual and was anxious to have the abdomen opened, as he had been working under great discomfort for over a year.

OPERATION (January 15, 1936).—The abdomen was opened through a right upper paramedian incision. An ulcer was found on the anterior wall of the

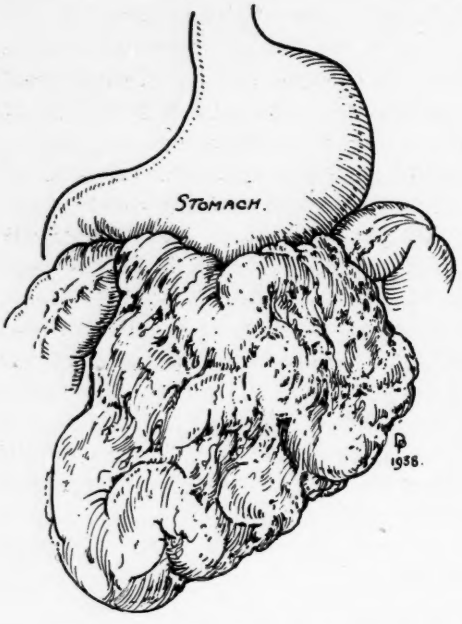


FIG. 667.—Showing the appearance of the lesser sac.

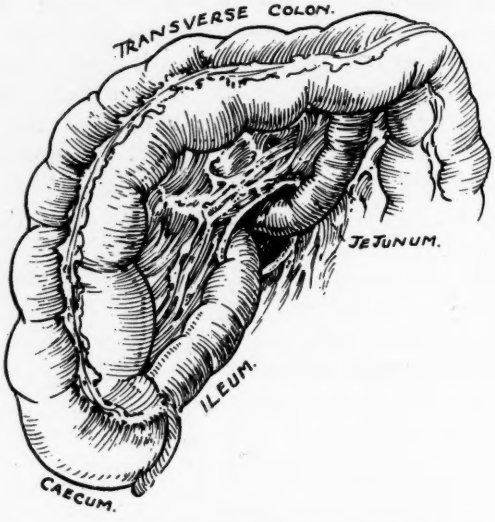


FIG. 668.—Showing the relationship of the contents of the sac.

duodenum, close to the pylorus. A posterior no-loop gastro-jejunostomy was carried out, using two rows of sutures. When I was about to put in a few points of suture, to anchor the junction to the aperture in the transverse mesocolon, I was reminded that a colleague of mine never put any in. Contrary to my invariable custom, I then did not insert any. The appendix was adherent and fibrous, and was removed. The abdomen was closed by two continuous catgut sutures and interrupted silkworm gut used for the skin.

SUBSEQUENT PROGRESS.—The progress of the case is interesting. On January 17 he developed mild bronchitis and also had hiccups for about eight hours. On January 18 he had subacute tonsillitis, which lasted until January 23. The temperature varied from normal to 100° F. during this period. The patient was quite well otherwise until January 28, when a slight discharge of blood-stained

fluid occurred from the middle third of the incision, and the next day a tag of omentum appeared.

The patient was taken into the operating theatre for resuture of the abdominal wall. It was found that the catgut had almost completely disappeared. After separating the omentum from the edges of the incision the small intestine was found to be in the omentum. On further investigation it was seen that the whole of the jejunum and ileum had entered the lesser sac through the opening in the transverse mesocolon, and had filled the lesser sac to its anatomical limits (*Fig.* 667). On lifting up the omentum and its enclosed intestine, the exact relations of the parts entering the opening could be made out. *Fig.* 668 gives a good idea of these relations. Fortunately there was no difficulty in pulling out the intestines. The necessary sutures were inserted to prevent a recurrence, before closing the abdomen.

Convalescence was uneventful, and the patient left hospital on February 12, 1936. He remained quite well until he left the district in July, 1937.

Comment.—This case is interesting because there were no symptoms, except possibly the hiccups on the day after the operation, to suggest that anything unusual had happened in the abdomen. If the catgut had not been absorbed rather too soon, it is possible that the hernia would never have been discovered. It makes one wonder if the frequency of this complication has not been underestimated in the past. Moynihan[1] mentions 4 cases, including one of his own, in which intestinal obstruction had supervened; also another case of his own, in which a subsequent operation accidentally revealed an internal hernia. He states that "all forms of hernia can be prevented by attention to two points: (1) The suture of the edges of the opening in the mesocolon to the stomach, or, preferably, to the jejunum; (2) The avoidance of a loop of jejunum between the duodeno-jejunal flexure and the anastomosis".

REFERENCE

[1] *Abdominal Operations*, 4th ed., 1926, i, 211. London: W. B. Saunders & Co.

BURST ABDOMEN

? A CURIOSITY

By H. B. WALKER, Minehead

The following case may justify record as a clinical curiosity:—

An urgent message was received to the effect that a man had 'burst his stomach'. Half an hour before, after coming in from a walk he had felt sick, retched, and felt a severe pain in his stomach. He was helped upstairs by his son. The patient was a retired dentist (unqualified), aged 58, a thin, pale, chronic alcoholic. He was lying on his back and supporting in his cupped hands over an exceedingly dirty pair of woollen drawers a large mass of entrails. To make asepsis still more complete these were covered over by a square of dirty handkerchief. His general condition was good, and he was comfortable. Mildly under the influence of alcohol, he was slightly jocular about his condition.

He gave a history of having had an operation for 'gastric ulcer' eleven years ago, and four years after that another one for perforated ulcer. The hernia which

subsequently developed had been getting worse for the last two years. He had not worn any supporting belt.

The bowels were gently wrapped in a clean towel and supported by a binder. It was noted that any undue pressure caused pain. He was given $\frac{1}{4}$ gr. of morphia and $\frac{1}{100}$ gr. of atropine, and removed to hospital.

About one and a half hours after the 'burst' the abdomen was repaired under gas and oxygen anæsthesia. The bowel mass, consisting of most of the small gut, was cleansed with saline. The skin rupture of about 4 in. long had taken place in the lower part of a supra-umbilical left paramedian scar about 8 in. long. The skin involved was thin and tore easily, like sodden brown paper, the whole length of the scar. The neck of the hernial sac was formed by the linea alba and the edge of the left rectus, and was 6 in. long and 3 in. across at its widest part. A knob of liver, entirely covered with omentum, bulged through the upper part of this orifice, to which it was roughly moulded. The general abdominal cavity was well shut off by omental adhesions. Such of these as was necessary for the replacement of the bowel were cut; redundant skin and weak fascial tissue were removed, and a closure and diminution of the sac was effected by utilizing the stronger tissues nearer to its neck.

No attempt at a 'radical cure' was made, but by overlapping and reinforcement as the conditions allowed, a substantial parietal wall was reconstituted. The temperature took about ten days to settle down, but otherwise convalescence was uneventful.

REVIEWS AND NOTICES OF BOOKS

Surgical Treatment. A Practical Treatise on the Therapy of Surgical Diseases. By JAMES PETER WARBASSE, M.D., F.A.C.S., Special Lecturer in the Long Island Medical College, and CALVIN MASON SMYTH, jun., B.S., M.D., F.A.C.S., Assistant Professor of Surgery in the University of Pennsylvania Graduate School of Medicine. Second edition. 9½ × 6¼ in. In three volumes, and an index volume. Pp. 2486, with 2237 illustrations. 1937. London: W. B. Saunders Co. Ltd. £7 7s. net.

THESE three volumes are well printed and handsomely bound, and much of the text is clear and attractive; the work shows the authors' immense industry. We regret that we are able to give only the mildest of praise, for both the text and the illustrations are of very uneven quality. Perhaps the very high standard of the surgical output of the Mayo Clinic has made us too exacting, but we feel that many of the illustrations in this work are neither useful nor ornamental.

The section on anæsthetics is well written and quite up to date; the same may be said of the chapter which deals with amputations and the fitting of artificial limbs; the photographs depicting these "amputés" at work are of great interest to every surgeon who has had experience of the surgery of war. The article on the Economic and Social Aspects of Surgical Treatment is one of the best in the three volumes; it is original, and shows, we should imagine, the trend of medical opinion towards a whole-time state service in the United States.

We shall proceed to point out some statements which, we venture to think, do not fairly represent modern surgical teaching. In the first volume we read with unaffected surprise that "phosphorus necrosis should be prevented by sparing human beings the necessity of working for a living at making phosphorous matches for somebody else's profit". This picture of the exploitation of the poor worker by the overfed financier would excite more sympathy, were it not the fact that in this country the disease, phossy jaw, has been extinct for the last forty years; every dental student knows that this was brought about by the substitution of red phosphorus for the dangerous yellow variety. Moreover, we believe that this little piece of chemical research was actually financed by the maligned capitalist.

The danger of omitting any reference to pathology in a surgical treatise is clearly shown in the treatment of scurvy and rickets; not a word is said about these being deficiency diseases; indeed, there is no mention of 'vitamins' in the index. A similar example of this danger is to be found under the treatment of Paget's disease of the nipple; this disease, they say, should be "promptly extirpated"—is the reader to infer that excision of the nipple is all that is necessary?

Few English surgeons would be found to agree that dislocation of the internal semilunar cartilage occurs "as the result of catching a floating body against the cartilage", or that the treatment of choice is the stitching of the loose cartilage to the capsular ligament; or even that "the results of this operation are good". This method was tried extensively many years ago and the results were most disheartening.

In Hodgkin's disease the early and complete removal of the enlarged glands is advocated; an extensive dissection of the neck must be carried out and, if they are adherent, the internal jugular and subclavian veins may have to be "sacrificed". The reader is not informed that such operations are of historical interest only and that surgeons of to-day content themselves with the removal of a single gland for microscopical confirmation. If the authors really consider that the wholesale removal of lymphadenomatous glands is the correct procedure, they should surely point out that they are almost alone in their opinion; we refuse to think that the compilers of this monumental system ceased to follow the trend of medical thought nearly thirty years ago.

Under the treatment of ascites due to cirrhosis of the liver they discuss the feasibility of diverting the portal blood into the inferior vena cava; this is, of course, the well-known physiological experiment of Eck's fistula, though they suggest making a lateral anastomosis,

whereas Eck divided the portal vein and put the afferent end into the vena cava. To the technique of this operation they devote nearly two pages; they devote some space to the difficulties, but say nothing of the dangers of this formidable proceeding in a patient poisoned with alcohol; nor do they mention that the experimental animals developed uræmic symptoms when fed on meat. The reader is left in doubt as to whether this method of treatment is seriously advised, for, in the second volume, they mention operations performed on men by Vidal and Rosenstein, whilst in the first they state that, as far as they know, this method has not been applied in "human surgical therapeutics". They acknowledge that Kocher described it as an unthinkable procedure, and at once go on, in the next line, to add "the surgeon should not attempt the operation until he has practised it on the cadaver". Indeed, their attitude to the subject of blood-vessel surgery seems to us to be unduly optimistic, for they affirm that the ideal treatment of aneurysm of the renal artery is by aneurysmorrhaphy.

When we arrived at the section on urology we rubbed our eyes to make sure we were not dreaming; it seemed almost as though we were transported back to the times of Frère Jacques and the itinerant lithotomists. Under the heading of lateral perineal cystotomy we found a detailed description of lateral lithotomy; nothing was missing—the neck of the bladder and the prostate are to be cut and, if necessary, a lithotrite is to be introduced through the wound in the perineum. We looked earnestly, but without success, for any hint that this method of removing a stone from the bladder is merely a matter of history, and somewhat remote history at that; very few surgeons in Great Britain have ever seen this operation done. In fairness to the authors we hasten to add that suprapubic cystotomy is offered as an alternative, but we think that the assertion that "the suprapubic operation is usually to be preferred to the perineal operation" might well have been more vigorously expressed.

After this, our special wonder is scarcely excited by the advice to pass a "seeker" to ascertain the presence and nature of a vesical calculus, nor by the instruction to wash out the bladder through a soft catheter before passing the cystoscope. We were, however, a little intrigued by the figure on page 188 of the third volume; this purports to show that the distended bladder, like the pleura, is to be emptied by a negative pressure apparatus; on this side of the Atlantic our difficulty in such cases is to ensure that the urine does not escape too quickly.

The study of these volumes strengthens our opinion that the day of these vast works is nearly over; few surgeons since Erichsen—and he enlisted Beck to help with the pathology—are such masters of surgery as to be in a position to review adequately the whole field, and composite works by numerous authors invariably lack continuity and are often far from consistent. We feel sure that the surgeon of the future will turn more and more to monographs.

If Dr. Warbasse and Dr. Smyth had omitted much that is useless and archaic from their *magnum opus*, the contents would probably fill about one volume and their reputation as surgical authors might have been enhanced.

The Science and Practice of Surgery. By W. H. C. ROMANIS, M.A., M.B., M.Ch. Cantab., F.R.C.S. Eng., F.R.C.S. Edin., Surgeon and Lecturer on Surgery, St. Thomas's Hospital, etc.; and PHILIP H. MITCHINER, M.D., M.S.(Lond.), F.R.C.S.(Eng.), Hon. Surgeon to H.M. The King; Hunterian Professor, Royal College of Surgeons of England; Surgeon and Lecturer in Surgery, St. Thomas's Hospital, etc. Sixth edition. 9¾ × 6¼ in. Vol. I, General Surgery. Pp. 808 + Index, with 393 illustrations and 13 plates. Vol. II, Regional Surgery. Pp. 978 + Index, with 337 illustrations and 6 plates. 1937. London: J. & A. Churchill Ltd. 14s. net each volume.

The Science and Practice of Surgery, in two volumes, by Romanis and Mitchener, has reached the sixth edition in the space of ten years. The rapidity with which each new edition has found favour is an indication of the merit of the work.

Authors of text-books on modern surgery have a difficult task in covering the whole field, yet comprehensive publications are more essential to the student now than ever before. In these days it is too often forgotten that the human body is a single complex machine, and that so far as surgery is concerned one organ or system must be considered in the light of its connections with others. Thus disorders in one region may depend upon other disorders far remote. There may be, on the other hand, multiple independent lesions

which must not be ignored or missed because one is more prominent than the others. For these and many other reasons the surgeon requires a wide knowledge and outlook. The volumes under review co-ordinate and describe in a rational manner the common problems with which the general surgeon should be familiar. The book is intended primarily for the education of the student and the enlightenment of the practitioner, but the teacher also will find inspiration if he turns over the pages before the delivery of a lecture.

A quotation is given in the preface to the first edition, which bears repetition: "Men who, led to take up surgery by the hope of gain and notoriety, have given themselves no time to learn its scientific principles; showy operators who diagnose with the knife and endeavour to dictate to nature and not to assist her". A text-book such as Romanis and Mitchener have written can do much towards the elimination of such men from the surgical field. Thus a good operator may be a bad surgeon, but the authors spoil the point to some extent by laying emphasis on exploratory abdominal procedures. The modern surgeon has at his hand the means of making an accurate pre-operative diagnosis in most cases. The necessity for exploration and the handling of innocent organs should seldom arise. The descriptions of various abdominal lesions with their diagnosis and treatment are accurate and to the point. The credit due to Moynihan and his immediate predecessors in the Leeds school is omitted, and his name does not appear in the index. Sir Robert Jones's methods and teaching are advocated to a large extent throughout the section on injuries and diseases of the bones and joints. Many illustrations are given which are modified from his works on orthopædic surgery. His name, however, seldom appears, and it also is omitted from the index. The authors wisely decided not to stress the methods of Böhler in the treatment of fractures. These methods may result in failure or disaster in the hands of those who have not the facilities found in hospital practice. It is pointed out in the preface that the so-called modern methods of treatment of fractures are in reality old. The reviewer treated fractures by the ambulatory method in plaster-of-Paris thirty years ago, a method which was adopted at that date in the hospital to which the authors belong.

Here and there throughout the two volumes there is a lapse from the high standard at which the authors have obviously aimed. For example, the statement that a blood-urea of over 40 mg. per 100 c.c. contra-indicates an immediate first stage prostatectomy must be regarded as inaccurate. A much higher blood-urea may be present without any loss of renal function. Again, the old Freyer operation is advocated for the removal of the prostate gland, and the statement is made that the Thomson-Walker method is "attended with great shock". This is by no means the experience of most operators. The question of catheter drainage, which is regarded by many surgeons as of first importance, is almost ignored. The description of the treatment of ruptured urethra follows the teaching of the old text-books; modern methods receive only casual mention. In a text-book covering the whole field of surgery space does not permit of great precision or of minute details, but such a statement as "proximal ligature is clearly impossible", in cases of innominate aneurysm, overlooks the work of Ballance and others, who found that the artery is free at its origin and that it could be ligated with success.

The opening chapters in Volume I are beyond reproach. Inflammation, Wounds, and Sterilization are admirably described.

Taken as a whole the book is a valuable contribution to current surgical literature.

Kleine Chirurgie. By Professor Dr. HANS KURTZAHN, Königsberg. Fourth edition. 8¾ × 6 in. Pp. 468 + viii, with 177 illustrations and a coloured plate. 1937. Berlin and Vienna: Urban & Schwarzenberg. RM. 12; bound, RM. 13.50.

THE fourth edition of this well-known German text-book of minor surgery is little changed from the third edition. Chapters on technique, anæsthesia, bandaging, and plaster work are followed by more detailed accounts of the treatment of injuries of soft parts, bones and joints, infections, tumours—benign and malignant—of the skin and subcutaneous tissues, more important special operations, and deformities. A final chapter gives advice on the assessment of disabilities, etc., due to accidents. The book gives an excellent idea of modern practice in the casualty ward and out-patient department of the German surgical clinics. It is well illustrated.

Précis de Diagnostic chirurgical. Published under the direction of Professor CH. LENORMANT, Paris. In four volumes. 7¾ × 5¼ in. Vol. I. General, by CH. LENORMANT and J. SÉNÈQUE. Head and Neck, by P. WILMOTH. Pp. 764, with 196 illustrations. Fr. 80; bound, Fr. 105. Vol. II. Thorax, Breast, Abdominal Wall, External Genital Organs, by J. PATEL. Pp. 490, with 138 illustrations. Fr. 50; bound, Fr. 70. Vol. III. Abdomen and Pelvis, by G. MÉNÉGAUX. Pp. 964, with 212 illustrations. Fr. 100; bound, Fr. 125. Vol. IV. Extremities, by J. SÉNÈQUE. Pp. 630, with 168 illustrations. Fr. 70; bound, Fr. 90. 1937. Paris: Masson et Cie.

THESE four small volumes constitute a guide to the modern French surgical practice of differential diagnosis. They make no claim to be complete in those special fields where the general surgeon rarely wanders, but are written for the student and young surgeon who wish to have a useful guide through the complexities of surgical diagnosis.

The first part of Volume I is devoted to general principles, injuries, surgical infections, diseases of the muscles, bones, and joints. The second part contains the special section on surgical affections of the head, neck, and vertebral column, but without reference to the special sense organs or the brain. The thorax, abdominal wall, and lower genital tracts are considered in the second volume. The largest volume (963 pages), like the previous two, is arranged on the topographical divisions of the region, which in this volume is the abdomen and pelvis, but there is an interesting chapter on six particular conditions: (1) Jaundice; (2) Post-operative pain; (3) Hæmorrhages from the digestive tract; (4) External fistulæ; (5) Gastric syndromes of infants, and (6) Foreign bodies in the intestinal canal. Each is discussed in full, but without too much bewildering detail. They are models of lucid writing and logical deduction. In the last volume the surgery of the limbs is considered, again on an anatomical basis.

The authors are to be complimented on the way in which they have followed the great French clinical tradition, not slavishly by ignoring modern diagnostic methods—chemical, radiological and bacteriological—nor by over-emphasizing these methods to lessen the importance of accurate clinical examinations and observations. The logical way in which the subject matter has been arranged and the clarity of the writing combine to make these volumes an excellent and attractive introduction to clinical surgery. The illustrations are numerous and well reproduced. Photographs and radiographs of the conditions discussed form the bulk of the figures.

St. Bartholomew's Hospital Reports. Edited by G. EVANS, G. GRAHAM, G. HADFIELD, C. F. HARRIS, W. SHAW, A. WORMALL, W. G. BALL, J. P. ROSS, and J. MAXWELL. Vol. LXX. Demy 8vo. Pp. 381 + xxviii. Illustrated. 1937. London: John Murray. 21s. net.

WE HAVE had occasion in previous years to draw attention to the sympathetic treatment of the biographical notes of the Hospital and School Staff on their decease by present members of the Staff or their successors; and the present volume is no exception to this rule.

Mr. Girling Ball is peculiarly happy in writing of the late Mr. Just, and no one could wish for a more sympathetic biographer. A very charming personality in the person of Professor Kettle is most generously dealt with, and Kettle's qualities as a man are brought out no less attractively than his eminence as a pathologist. To the surgeon, one of the most instructive papers is that of Messrs. Burrows and Coltart on treatment by manipulation. It is a very fair résumé of what can be done by this method. Clinicians will find interest in Mr. Ball's account of his own attacks of inflammation of the gall-bladder, which deviated very much from written authorities on this subject, and will be afforded an opportunity of judging how far they coincide with their own experience and how far they are abnormal. Dr. Harris records 37 cases of pyloric stenosis and their treatment in a paper which advocates a more general recourse to surgery; and Mr. Keynes contributes a thoughtful account of Rammstedt's operation as performed in recent years at St. Bartholomew's.

Surgical Anatomy. By GRANT MASSIE, M.B., M.S.(Lond.), F.R.C.S. (Eng.), Assistant Surgeon, Guy's Hospital; Lecturer in Operative Surgery, Guy's Hospital Medical School. Third edition. 9½ × 6¼ in. Pp. 468 + x, with 153 illustrations, many in colour. 1937. London: J. & A. Churchill. 18s. net.

THAT a third edition of Grant Massie's *Surgical Anatomy* should be necessary within three years of the second is abundant evidence that it fulfils a want amongst the literature available

for a student's education. It would be interesting to see what new point a student would take up if he founded his anatomical training on such a text-book and read no other, for it seems to contain all the facts of topographical anatomy which a student should possess as a foundation for his surgical training. The diagrams are, as a whole, good in their depiction of typical parts, and the book is well got up.

The Abdominal Surgery of Children. By Sir LANCELOT BARRINGTON-WARD, K.C.V.O., Ch.M., F.R.C.S. (Eng. and Edin.), Surgeon to H.M. The King; Senior Surgeon, Hospital for Sick Children, Great Ormond Street; Senior Surgeon, Royal Northern Hospital, London. Second edition. Demy 8vo. Pp. 333 + xv, with 97 illustrations and 4 coloured plates. 1937. London: Oxford University Press. 25s. net.

THIS is the second edition of a book which has come to occupy an important place in the literature of pediatric surgery. The surgery of childhood differs in many respects from the parallel entities of adult life, and recognition of this truth has led to the establishment of children's hospitals and the pursuit of pediatric surgery by individuals who in virtue of training and inclination are attracted to this branch of work. Sir Lancelot points out that, if we accept the distinctions of pediatric surgery as a whole, the principle applies in particular measure to abdominal surgical affections of childhood. This book is intended to present the considerations of this special branch of children's surgery, and it will be agreed that the author has fulfilled his task in a most effective and satisfactory manner.

The introductory chapter deals with what are called "General Principles", such features as the peculiarities of manifestations of disease in the child, the psychological equipment of the pediatric surgeon, and the reaction of the sick child to the treatment which he receives. In this last connection the author has done good service by drawing attention to the serious disadvantages, and it may be the actual risks, associated with pre-operative purgation and starvation. It is recommended that no purgatives or enemata be given the day before operation, and the usual diet should be permitted until the morning of the operation, barley sugar being given in abundance in the meantime.

When the condition of the patient or the gravity of the operation demands special care, additional precautions are taken to increase the resistance of the patient, such as the administration of saline-glucose or blood transfusion, but with these exceptions the routine is on the simple lines which have been indicated. Attention is drawn to the improvement which has followed this régime, and a particular point is made of the fact that since its adoption no fatal case of post-operative acidosis has occurred. The author attributes this happy state of affairs to a re-adjustment of the preparatory routine, and this may well be so, but it should be noted that chloroform followed by open ether was the anæsthetic employed when acidosis developed.

The volume includes consideration of diseases of the abdominal wall and such related conditions as inguinal hernia. This section is presented in an attractive and exhaustive way, but the pitfalls of re-edition are evident when one reads on page 35 in relation to the descent of the testis "Little is known of the nature of the forces which cause the testis to migrate . . .", while on page 55 the same problem is discussed at considerable length.

There seems to be some confusion as to the treatment recommended in pneumococcal peritonitis. It is stated that "surgery offers the best hope, and the sooner it is employed the better", but immediately afterwards the context reads "of the Great Ormond Street cases none was operated on within four days of the onset. Operation at the height of the septicæmia is as hopeless as is an operation for empyema in the course of a raging pneumonia". It is difficult to reconcile these statements, and it leaves the reader uncertain as to the course which the author recommends.

These, however, are but minor criticisms; the main theme of the book is excellent, and in this, its second edition, it has been brought up to date in a most commendable manner. There is no doubt that certain parts of the book are better than others; the chapter on the vermiform appendix, for example, deserves special praise, for it is written in a most attractive style, maintaining throughout the hall-mark of practical experience. In contrast to this, the section on intestinal obstruction is disappointing; reference to recent experimental work is scanty, and for this reason the considerations of treatment are somewhat inconclusive.

Criticism, however, is always easy, and no book is immune; in this instance there is no ground for any serious criticism, for it is a contribution of practical value, and every page

indicates that it is the work of one who has first-hand knowledge of his subject. The illustrations are numerous and informative, while the type and paper make reading an easy and a pleasant task.

Appendicitis. A Clinical Study. By W. H. BOWEN, Hon. M.A. (Camb.), M.S. (Lond.), F.R.C.S., Hon. Surgeon to Addenbrooke's Hospital, Cambridge; Examiner in Surgery, University of Cambridge, etc. With an Introduction by Sir ARTHUR HURST. Crown 8vo. Pp. 202 + xii. 1937. London: Cambridge University Press. 7s. 6d. net.

A CYNICAL reviewer might urge as a reason against the writing of a new book on this subject that there has been nothing startling or revolutionary published since the article by Sir David Wilkie in 1914. We think this idea is erroneous; a well-written monograph, even though it contain no obvious departure from the beaten track, is always welcome. It will serve to refresh the reader's memory, may stimulate him to review his own cases, and even, perhaps, raise in his mind the uneasy feeling that he may at times have committed faults of both commission and omission.

The author has had a wide experience of this disease; he has kept careful notes and has "intended his mind" on them; he is intensely interested in the pathology of the appendix and he has kept abreast of the literature: no wonder that the book is a success.

We congratulate him on the avoidance of two pitfalls. The cases he quotes are incorporated in the text and are not printed in italics, and his references are to be found at the bottom of the page. It is to his credit that they are mostly to English writers and, when possible, to authors of his own medical school.

He has taken much trouble in looking up cases at various periods both before and after the advent of the surgeon in the history of appendicitis; his contention that this disease is not only much more frequent but of a more severe type seems to be amply proved. He has been impressed, and possibly startled, by the low mortality when appendicitis was treated in the medical wards of Guy's Hospital; his explanation of this is that the physicians not only prescribed absolute rest but gave morphia as a routine. When surgery came on the scene, morphia was at once taboo and purges were sometimes ordered.

The analysis of many cases leads to the conclusion that the presence of concretions and the unwise giving of purges are the two main factors in setting up a diffuse peritonitis; he gives good reason for denying that constipation plays any part in causing or aggravating the disease, and he resolutely sets his face against the presence of kinks and bands as playing any part in the causation.

He is emphatic on the importance of the history of the onset; to start with, the pain is a vague stomach-ache of para-umbilical or transverse abdominal localization; only some hours later does it move to the right iliac fossa; with the history of a pain starting in the right iliac fossa, he would hesitate to make a diagnosis of appendicitis and would reconsider the symptoms before making up his mind.

He favours opening the abdomen at the outer border of the right rectus—Battle's incision. Whilst this route does lead directly to the base of the appendix, we think many surgeons will find it difficult to avoid, as Mr. Bowen does, damaging at least part of the nerve-supply to the muscle; an energetic assistant may easily tear one of the nerves by his retraction. It might be wiser, when there is any doubt, to divide one nerve deliberately and suture it later; this will double the space at the disposal of the operator; the rectus has several other motor nerves, and, after all, what is a partial paralysis of an abdominal muscle to the risk of rupturing a distended appendix?

The author's interest in the appendix does not cease with the end of the operation; he has some interesting remarks to make on its behaviour after it has been removed; he may almost be said to have extended the pathology of the living to that of the still just alive.

When the second edition is called for, we should value his considered opinion on the condition known as ileus duplex; we should also like to know what he thinks of opening a pelvic abscess through the rectum, which we have seen followed by some dramatic recoveries. Perhaps his answer would be that this complication should not be allowed to occur.

The book has been written with great care and the style is simple and lucid; withal, there is a faint but persistent aroma of the archaic. After much reflection, we hazard the guess that the author has, perhaps unconsciously, modelled his prose on that of the late Mr. Jacobson; even so, he has hitched his wagon to a star. Even at this distance, we tremble

to think what the scholarly but irascible author of *The Operations of Surgery* would have exclaimed if he had found a favourite dresser using the word "fossa" as though it were a plural (p. 157), and writing of "a phenomena" (p. 126). We apologize for pointing out errors in the classics to a surgeon at one of the more revered of our Universities and should like to think that the lapsus in each case is to be blamed on the printer.

We fully agree with Sir Arthur Hurst, who writes in his delightful introduction that this monograph is the best review of the subject in the English language; if we refrain from discussing the book in more detail, it is because we feel that it will be read by every surgeon who is keen on keeping his knowledge up to date.

Mr. Bowen has upheld the Guy's tradition of clinical investigation, and is an outstanding example of an old adage from Indian medicine: "He who combines reading with experience proceeds safely and surely like a chariot on two wheels."

Tumours of the Nervous System. An Investigation of the most Recent Advances. Being The Proceedings of the Association for Research in Nervous and Mental Disease, New York, December 27 and 28, 1935. 9¼ × 6 in. Pp. 493 + xxii, with 213 illustrations. 1937. Baltimore: Williams & Wilkins Co. (London: Baillière, Tindall & Cox.) 34s. net.

As the title suggests, the book is concerned with the pathology of many of the tumours of the nervous system, in particular the gliomas, ependymomas, meningiomas, and tumours of the peripheral nerves. Chapters, however, are devoted to more clinical considerations. The diagnosis by pneumo-encephalography of tumours arising in the corpus callosum is discussed by R. W. Waggoner and Konstantin Lowenberg; the assessment of the value of changes in the chemistry and cytology of the cerebrospinal fluid by Fremont Smith; and a most interesting chapter is written by Elsberg on a recent attempt to measure the finer changes in the olfactory sense, which, he believes, will soon occupy a position equal in importance to that of perimetry in the localization of a cerebral tumour.

The book opens with a chapter written by R. G. Canti, J. O. W. Bland, and Dorothy Russell as a commentary on the tissue-culture film made by Dr. Russell with the collaboration of the other two writers, one of them most unhappily now dead. Astrocytoma, oligodendroglioma, and spongioblastoma multiforme were shown in culture. One of the most impressive points demonstrated was the difference between the astrocytoma and oligodendroglioma cells in vigour of movement as well as in form, showing that they are of truly independent cell type, although they may be mixed in the same tumour. This is a pioneer effort in clarifying the neuroglia problem by a different method. Dr. Louise Eisenhardt contributes a most encouraging follow-up of tumours with long survival periods, showing that even the more malignant gliomas, the glioblastoma and medulloblastoma, can be brought under control for periods of economic value. One patient from whom a cerebellar astrocytoma was removed is alive 28 years later; another, who had a cerebral astrocytoma, 21½ years later; and a third, with a pituitary adenoma, 24 years after operation.

On the more academic side the results of work on the problems of cerebral tissue metabolism are stated in three short chapters. The sensitivity of cerebral tumours to radiation is discussed on the basis of histological changes following adequate treatment. Penfield and his co-workers, Elvidge and Cone, review a series of 210 proved gliomas (which form 44·9 per cent of their total collection of tumours of the central nervous system). They use the classification of Percival Bailey, the only alteration being a subdivision of the astrocytomas into three groups: pilocytic, gamistocytic, and astrocytoma diffusum. This paper gives one of the clearest statements extant of the criteria on which histological diagnosis should be made. Globus has done something new. He has formulated a new classification of the meningiomas based on the autogeny and phylogeny of the three covering membranes of the brain. His thesis depends on the belief that the pia, arachnoid, and dura mater all develop from the same primordial elements: from a single mature element or from a combination of two or all three subdivisions or splittings which are to form the various covering membranes. The idea is contrary to the established belief that meningiomas arise in the arachnoidal tufts, and is not in harmony with the cyto-architectural classifications of Bailey and Bucy, which have been of such great service in the past. The Kernohans review a series of 109 ependymomas, of which 54 were in the spinal cord or filum terminale, 52 in the cranial cavity, and

3 in the hollow of the sacrum. They give a new classification, including the choroid plexus papillomas as a variant. Leo Alexander discusses the development of the lateral recess, and removes many of the difficulties in the understanding of the pathology of cysts and solid tumours which grow in the cerebellar-pontine angle. The book closes with two very important papers on tumours of the peripheral nerves.

This is a volume which any critical reader can peruse with benefit and pleasure.

The Collapse Therapy of Pulmonary Tuberculosis. By JOHN ALEXANDER, B.S., M.A., M.D., F.A.C.S., Professor of Surgery, University of Michigan, etc., with contributions by MAX PINNER, JOHN BLAIR BARNWELL, and KIRBY SMITH HOWLETT, jun. 10½ × 7½ in. Pp. 705 + xvi, with 367 illustrations. 1937. London: Baillière, Tindall & Cox. 67s. 6d.

THIS is a younger, but how much more stalwart, brother of the author's well-known *Surgery of Pulmonary Tuberculosis*, published in 1925. It is a comprehensive, balanced account of the subject, based on the author's wide experience. Some of the material of the older book, especially the history of collapse therapy, has been reproduced in modified form, and it is pleasing to see that the portrait gallery of pioneers in this branch of surgery has been retained.

The first chapter gives many statistics to justify the conclusion that "the results of unaided sanatorium treatment in the far-advanced stage of tuberculosis and in certain types of the moderately advanced stage are shockingly bad. Collapse therapy . . . has tremendously improved the results in these stages and has also bettered the results in the minimal stage."

The physiology and pathology of pulmonary collapse are discussed. There is a valuable chapter on the choice of operation, with a remarkable series of eighty-four line drawings, describing lesions and the suggested possibilities of treatment. The indications, contra-indications, technique, and management of the different methods of collapsing the lung are described in detail. The chapters on the modern thoracoplasty operation, in the development of which Professor Alexander and his co-workers have taken so important a share, are among the best. European readers will be surprised that in the section on extrapleural pneumothorax, no mention is made of the recent work of Graf, of Dresden, and Schmidt, of Heidelberg. Carl Semb and his now well-known operation receive no specific mention in the text, though there is a reference in a footnote on page 378. The bibliography and the text had obviously been completed before the arrival of tomography.

The X-ray reproductions and other illustrations are pleasingly clear, and the whole volume worthily maintains the reputation of the great school of thoracic surgery of which Professor Alexander is the head. It will naturally find its way into the library of every thoracic surgeon, but it is to be hoped that the book will be widely studied by internists, for whom, as well as for surgeons, it is stated by the author to have been written.

Las Neumectomias totales. By Dr. JORGE A. GALARCE. 9⅛ × 6¼ in. Pp. 309, with 157 illustrations. 1937. Buenos Aires.

THIS thesis does not pretend to the status of a complete monograph, because the author indicates that he has not the personal experience upon which to found a treatise, but it does, as he intended, provide a very useful résumé of the subject and its literature.

In a first part the author relates the history of pneumonectomy, gives an account of the anatomy and physiology of respiration, and relates the results of experimental operations.

In the second part he sets out the indications for removal of the lung; the diagnostic methods; pre-operative preparation, including artificial pneumothorax; anæsthesia; the technique of operation; post-operative treatment and complications; and finally, forty-five pages of description of cases from the leading thoracic surgeons.

A book of reference useful to those interested in this branch of surgery. Tudor Edwards, Roberts, Magill, and other British contributors to the advance of thoracic surgery receive a proper share of attention.

Primary Carcinoma of the Lung. By EDWIN J. SIMONS, M.D. With a Foreword by J. ARTHUR MYERS, M.D., Professor of Medicine and Preventive Medicine and Public Health, University of Minnesota, Minneapolis. 9 × 6 in. Pp. 263, with 30 illustrations and 2 coloured plates. 1937. Chicago: The Year Book Publishers Inc. (London: H. K. Lewis & Co. Ltd.) 21s. net.

THE opening words of the author's summary, "this compilation of cases, opinions, and facts", sufficiently indicates the character of this book. That it has been produced by a man practising in a village community of 435 souls, is a tribute to his industry. The bibliography runs to well over five hundred entries, and he can draw upon published records of over five thousand cases.

His analyses and tables cannot fail to be of value even to those whose special work lies in the field of thoracic surgery, and though to them the record of others' experience in matters of diagnostic and therapeutic technique cannot be of great help, to those whose interests are more general, it will prove a useful review of the situation as it stands to-day.

The evidence which proves the real increase in the incidence of bronchial carcinoma is here well marshalled. All that the author can say as to aetiology, of course, amounts to no more than that every source of 'irritation' of the pulmonary mucosa may be an antecedent of cancer. As to pathology, he indicates the occurrence of hilar and peripheral primary locations; with regard to histology, he indicates the settling of opinion as to the common origin of squamous, adenomatous, and oat-celled types, all from the basal cell of the bronchial epithelium.

Males suffer four times as frequently as females. As to the age incidence, 80 per cent of cases occur between 40 and 70, and 34 per cent of the whole in the sixth decade. The right lung is affected rather more frequently than the left; the left upper lobe a little more often than the right upper; both uppers more often than lowers. The frequency with which organs and tissues suffer metastases is tabulated. The symptomatology is considered statistically and the physical signs are classified. The illustrations are reasonably good. It is certainly a book that ought to be in every medical library.

The Lung. By WILLIAM SNOW MILLER, Emeritus Professor of Anatomy, University of Wisconsin. 9 × 6 in. Pp. 210 + xv, with 152 illustrations. 1937. London: Ballière, Tindall & Cox. 34s.

TO William Snow Miller more than to any other single investigator do we owe our knowledge of the intimate structure of the human lung. He was the pioneer worker on the detailed anatomy of the ultimate pulmonary air spaces, and his description of their arrangement has stood the test of over forty years. Only in one detail has there been any challenge to his description. Miller maintained that adjacent pulmonary alveoli communicated with each other only through their common openings into an "atrium". Other investigations have suggested that there exist pores or openings between neighbouring alveoli. Miller did not deny their existence, but showed that they must be regarded as the result of pathological processes.

The chapters on the blood-vessels, pulmonary and bronchial, on lymphoid tissue, and pleura are illustrated by original drawings, many reproduced in colour. An interesting chapter on the history of our knowledge of lung structure and a good bibliography conclude the book. This monograph can be recommended to all those who wish to make themselves familiar with normal lung anatomy.

Surgical Pathology of the Thyroid Gland. By ARTHUR E. HERTZLER, M.D., Surgeon to the Agnes Hertzler Memorial Hospital, Halstead, Kansas; Professor of Surgery, University of Kansas. 9½ × 6½ in. Pp. 298 + xviii, with 238 illustrations. 1937. Philadelphia and London: J. B. Lippincott Co. 21s. net.

THE author gives his conclusions on diseases of the thyroid gland after a long and extensive clinical experience, and, since this is a personal record, he omits any extensive reference to the literature or discussion of the experimental work on the aetiology. The text is lavishly illustrated with excellent photographs, but is marred by the flippancies which are becoming usual even in serious American books.

After describing the normal anatomy of the gland, an attempt is made to assess the significance of the changes due to advancing age. Of the four main types in the classification of the American Society for the Study of Goitre, he regards the first three—namely, (1) Non-toxic diffuse, (2) Non-toxic nodular, and (3) Toxic nodular goitre—as progressive stages in the same degenerative process, combined with the toxicity of degeneration, whereas the fourth type—toxic diffuse goitre (Graves' disease)—is a toxicity of proliferation. Even if a goitre does not become toxic in the accepted sense, he believes that the toxicity of degeneration will inevitably lead to a "cardiac death", and that the only logical treatment is total thyroidectomy. Since the normal gland undergoes degenerative changes identical with those seen at an earlier age in goitres, the author suspects that once the thyroid has brought us safely to the adult stage and starts its natural process of degeneration, it produces toxins which inevitably lead to death from heart failure. He does not, however, recommend wholesale thyroidectomy as a substitute for the philosopher's stone. Unfortunately the author gives us no evidence in support of his views, and there appears to be no reason for incorporating them in such an elaborate publication, other than that of completing his series of monographs on surgical pathology.

Schmerzen und Leistungs-störungen bei Erkrankungen der Wirbelsäule. By Dr. med. EDUARD GÜNTZ. 9¾ × 6½ in. Pp. 171 + viii, with 88 illustrations. 1937. Stuttgart: Ferdinand Enke. Paper covers, RM. 15.20.

IN this monograph Schmorl's pathologico-anatomical teachings are correlated with clinical symptoms. A discussion of important points in history and clinical examination is followed by a chapter dealing with some of the common pitfalls in X-ray diagnosis due to overlapping, distortion, etc. Amongst other valuable technical hints, the author stresses the importance of taking survey-films at a distance of no less than 130 cm. The reproduction of X-ray films well support the author's statements.

In adolescent kyphosis the absence of pain is emphasized, and it is stated that if pain does occur it is limited to regions of the vertebral column which are not affected by the primary disease. The pain is explained as an expression of an active, compensatory lordosis, and must be regarded as being of muscular and ligamentous origin. Similar conditions are found in spondylosis deformans, unless the process affects the cervical spine. In this region pain may be caused either by disturbances of posture and movement or more rarely by a narrowing of the intervertebral foramina producing direct pressure on nerves.

Osteochondritis of the intervertebral disc is regarded as a fairly common, painful disorder in which a history of relatively slight trauma is frequently obtained.

Lastly senile kyphosis and deposition of lime in the intervertebral disc are described.

Only a passing reference is made to the backward prolapse of the nucleus pulposus, which in recent years has become an important clinical entity, chiefly through the work of Mixter, Barr, and Ayer.

A number of illustrative cases have been selected from a large clinical experience, and they greatly add to the interest of the subject. The bibliography deals predominantly with the German literature.

Die angeborenen Fehlbildungen der menschlichen Hand. By Professor WALTHER MÜLLER, Königsberg. 10 × 6¾ in. Pp. 170, with 116 illustrations. 1937. Leipzig: Georg Thieme. RM. 14; bound, RM. 15.50.

THE author has set himself the task of describing the congenital malformations of the human hand on the basis of biological, i.e., developmental and genetic characteristics. He rejects the purely anatomical classification as misleading and inefficient. He recognizes: (1) Malformations as arising from defects of the primitive tissues destined to form the skeleton; and (2) Defects caused by a disturbance in the development of the primitive handplate, i.e., the tissues from which the soft structures of the hand are formed. These two developmental processes run parallel, but are governed by different genes. They are, however, closely interrelated.

The first part of the book is devoted to malformations caused by disturbances in the development of the skeleton-anlage. This includes numerical variations and abnormalities in length. The embryological approach leads the author to regard polydactylism

and oligodactylism as two anatomically different expressions of the same fundamental biological tendency.

The second part deals with disturbances of the primitive handplate. An insufficient differentiation here leads to such malformations as syndactylism and cleft hand. Hyperplasia of the primitive plate is the cause of circumscribed gigantism.

In the third and last chapter, abnormalities occurring at a later stage in development are discussed.

The hereditary and genetic characteristics are traced in all groups, and the author has certainly succeeded in furthering our understanding of a bewildering multiplicity of static anatomical entities by following a rational classification, and by a description in terms of development.

Siebzehn Jahre Strahlentherapie der Krebse. Zürcher Erfahrungen, 1919-1935. By HANS R. SCHINZ and ADOLF ZUPPINGER. 10 × 6¾ in. Pp. 340 + x, with 95 illustrations and 213 tables. 1937. Leipzig: Georg Thieme. RM. 32; bound, RM. 37.

THIS volume is the result of seventeen years' work in the Röntgen Institute of Zurich, and we would say at once that we consider it a document of the very first importance in the study of the treatment of cancer by radiation. Dealing, as it does, with 2529 cases of cancer in every region, it embodies the results of a very wide experience, but the value of this experience is immensely increased by the manner of its presentation. Quite apart from the subject matter with which it deals, a careful study of this volume would be well worth while for anyone engaged upon a clinical research. A vast amount of information of the most detailed kind has been brought together so skilfully that it can be studied with facility in minute detail, whilst for those who wish to obtain a general idea of the results, two abstracts, one detailed and the other occupying only two pages, give a perfect general view of the whole field which is covered.

The volume opens with a discussion of the incidence of carcinoma in the town of Zurich, so analysed as to satisfy the demands of the most exacting statistician. Whilst presenting many points of interest it leads to the general conclusion that the apparent increase of carcinoma is fully accounted for by the increasing longevity of the inhabitants.

There follows an account of the Röntgen Institute itself, and a full general account of the methods employed. In some ways this forms the most valuable section of the report, and we were particularly struck by the ingenious methods by which the cases are classified, and by which the radiation treatment is described and represented in tabular form. In the illustrative examples which are given, one not only sees at a glance the nature of the case and the result obtained, but one can grasp instantly the precise method of treatment adopted and the extent of radiation employed. Into simple charts and a few lines are condensed a mass of material. which without these methods would occupy many pages, and would exhaust without informing,

The bulk of the report follows upon this introduction, and deals in detail with the whole of the material which passed through the clinic in seventeen years. Illustrative cases are minutely described, and the whole of the cases are divided up into such small groups that for practical purposes one can study them as individuals. The cases are presented with the utmost candour, and failures are studied quite as carefully as the successes. Each group of cases is gathered together into a small table of a standard type, from which one can see at a glance the results obtained after one, three, or five years.

The report concludes with a detailed abstract in which the whole of the information is gathered together and elaborated in tables of exceptional clearness, whilst the final short abstract gives a bird's-eye view of the statistical material on which the report is based.

We do not know which to admire the more, the admirable work of which this is a report, or the excellent manner of its presentation. We are convinced that it will repay anyone interested in the subject of cancer to give the report the most careful study.

Short-wave Diathermy. By TIBOR DE CHOLNOKY, Associate in Surgery, New York Postgraduate Medical School, Columbia University. 9¼ × 6¼ in. Pp. 310 + xvi, with 38 illustrations. 1937. New York: Columbia University Press. (London: Humphrey Milford, Oxford University Press.) 20s. net.

AT the outset the author makes it quite clear that he makes no claims for short-wave therapy as a panacea for all ills. He pleads for a more tolerant attitude towards those who are

labouring to bring what he considers to be a most significant contribution to physical therapy out of the mists of uncertainty. In this book Mr. Tibor de Cholnoky gives a short and very comprehensive outline of short-wave diathermy, and those who have more or less discarded this form of treatment are recommended to read this work.

The book is divided into six parts. Part 1 gives in a few pages a brief outline of the history of short-wave diathermy. Part 2 deals with the physics of the subject; this part is well illustrated with clear explanatory diagrams. Part 3 discusses at some length the experiments on bacteria and other organisms, and experiments on animals. Part 4 gives a careful account of the technique; this is admirably done, and should be read with care by those who are proposing to undertake this form of therapy. Part 5 cites the clinical applications of short-wave diathermy. Most books of this kind give long lists of diseases in which the special treatment under discussion can be used, and this book is no exception. A good deal of the matter is rather speculative, and one feels that this section could be with advantage shortened. Many of the diseases for which short-wave diathermy can be used apparently only derive 'beneficial effects' from the treatment, which is perhaps another way of saying it does no harm. Again it seems unnecessary to allude to the possibility of treating diabetes mellitus by means of short-wave diathermy unless it is a safeguard against an improbability becoming a fact. However, due credit must be given to a treatment which is said to banish the common cold. Nevertheless the author makes no exaggerated claims, but considers the matter of the results of treatment impartially and with commendable restraint. Part 6 is the conclusion of the book, and is really a neat summary of the author's views.

The radiographs on pages 139 and 236 are not good examples to illustrate the effect of treatment. On page 139 the radiographs 139 *a* and *c* are not well produced, as in radiograph 139 *c* a good deal of the periphery of the thorax is cut off on the affected side, which does not make the example as an illustration of the effects of treatment a very convincing one. On page 236 the radiographs are not comparable, as radiograph *a* seems to be underexposed.

Apart from these criticisms the book is interesting to read and should be an asset to the physiotherapist's library.

BOOK NOTICES

[*The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest for our readers.*]

La Hernie postérieure du Ménisque intervertébral (Hernie nucléaire postérieure) et ses Complications nerveuses. By PIERRE GLORIEUX, Bruges. Imp. 8vo. Pp. 102 + viii, with 98 illustrations and 2 plates. 1937. Paris: Masson et Cie. Fr. 40.

Surgical Diseases of the Mouth and Jaws. By EARL CALVIN PADGETT, B.S., M.D., F.A.C.S., Associate Professor of Clinical Surgery, University of Kansas School of Medicine, Kansas City; Associate Professor of Oral Surgery, Kansas City Western Dental College. 9½ × 6½ in. Pp. 807, with 334 illustrations. 1938. London and Philadelphia: W. B. Saunders Co. Ltd. 45s. net.

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